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TWENTY-THIRD ANNUAL MEETING

OF THE

AMERICAN GASTRO-ENTEROLOGICAL ASSOCIATION

HELD AT THE

HOTEL TRAYMORE, ATLANTIC CITY, N. J.

May 3 and 4, 1920

Printed for the Association
St. Louis
1920

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Julius Friedenwald	1013 N. Charles St	Baltimore, Md.
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B. B. Vincent Lyon	1901 Pine St	Philadelphia, Pa.
Morris Manges	72 E. Seventy-ninth St	New York, N. Y.
Thomas C. Martin	1725 N St., N. W	Washington, D. C.
William J. Mayo	Mayo Clinic	Rochester, Minn.
George W. McCaskey	407 W. Main St	Fort Wayne, Ind.
Alexius McGlannan	114 W. Franklin	Baltimore, Md.
Wm. Edward McGuire	6 E. Franklin St	Richmond, Va.
Lafayette B. Mendel		New Haven, Conn.
Willy Meyer	700 Madison Ave	New York, N. Y.
Robert Talbott Miller	1226 Murray Hill Ave	Pittsburgh, Pa.
R. Walter Mills	3701 Westminster Place	St. Louis, Mo.
Wm. Gerry Morgan	1624 Eye St	Washington, D. C.
Fred T. Murphy	Washington U. Med. School	St. Louis, Mo.
Leo. H. Neuman	194 State St	Albany, N. Y.
Eugene L. Opie	Penobscot Bldg	Detroit, Mich.
Franz Pfaff	29 Gloucester St	Boston, Mass.
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Robert Pollock	1000 Watts Bldg	San Diego, Cal.
M. Milton Portis	104 S. Michigan Ave	Chicago, Ill.
Edward Quintard	145 Fifty-eighth St	New York, N. Y.
Martin E. Rehfuss	1934 Chestnut St	Philadelphia, Pa.
David Riesman	1715 Spruce St	Philadelphia, Pa.
Dudley D. Roberts	270 Park Ave	New York, N. Y.
Joseph Sailer	1718 Spruce St	Philadelphia, Pa.
George R. Satterlee	125 W. 58th St	New York, N. Y.
John P. Sawyer	536 Rose Bldg	Cleveland, Ohio
Sidney K. Simon	3439 St. Charles Ave	New Orleans, La.
Bertram W. Sippy	122 S. Michigan Ave	Chicago, Ill.
Frank Smithies	1002 N. Dearborn St	Chicago, Ill.
Horace W. Soper	3701 Westminster Place	St. Louis, Mo.
Charles D. Spivak	1421 Court Place	Denver, Colo.
Charles G. Stockton	436 Franklin St	Buffalo, N. Y.
Philemon E. Truesdale	151 Rock St	Fall River, Mass.
Fenton B. Turck	14 E. Fifty-third St	New York, N. Y.
Franklin W. White	322 Marlborough St	Boston, Mass.

DECEASED MEMBERS.

Samuel Fenwick	London, England
Adolph Kussmaul	Heidelberg, Germany
Franz Riegel	Giessen, Germany
D. D. Stewart	Philadelphia, Pa.
J. Dutton Steele	Philadelphia, Pa.
A. D. Kohn	Chicago, Ill.
Frank H. Murdock	Pittsburgh, Pa.
James P. Tuttle	New York, N. Y.
Jesse S. Myer	St. Louis Mo,
C. A. Ewald	Berlin, Germany
Henry L. Elsner	Syracuse, N. Y.
George D. Kahlo	White Sulphur Springs, W. Va.
Walter J. Dodd	Boston, Mass.
S. J. Meltzer	New York, N. Y.

CONSTITUTION AND BY-LAWS

OF THE

AMERICAN GASTRO-ENTEROLOGICAL ASSOCIATION.

CONSTITUTION.

ARTICLE 1. This Association shall be known as the American Gastro-Enterological Association.

ARTICLE 2. The object of this Association shall be the promotion of investigation of the normal and pathological conditions of the digestive organs.

ARTICLE 3. Membership in this Association shall be of three kinds: (*a*) Honorary, (*b*) Corresponding, (*c*) Active.

(*a*) Honorary membership shall be restricted to such as have attained an international reputation by their published works and have scientifically furthered the subject of gastro-enterology. Their nomination shall take place at the annual meeting, upon the written suggestion of at least six active members and on the recommendation of the Council of the Association at the next annual meeting. Their election shall be by unanimous vote of the members present.

(*b*) Corresponding membership shall be restricted to foreign scientists in recognition of meritorious work within the scope of the Association. Candidates for such membership shall be proposed by at least six active members and receive the recommendation of the Council at the next annual meeting. Their election shall be by unanimous vote of the members present.

(*c*) Active membership shall be restricted to American and Canadian investigators and practitioners who have published meritorious work in normal or pathological anatomy or physiology, in medicine or surgery of the digestive canal and its secretory appendages, and who enjoy an unimpeachable moral standing in the medical profession.

Names of candidates for active membership must be: (1) Proposed by two active members who are not members of the Council, such proposal to be submitted to the Committee on Admissions and Ethics, which should report to the Council as soon as possible; (2) Must be recommended by the Council and (3) must receive a majority of the votes of active members present at the annual meeting of the Association.

The election of active members shall take place at the annual meeting of the Association and shall be by ballot.

It shall be the duty of the Secretary to notify active members of all nominations that are to be submitted to the Association at the annual meeting.

Article 4. The officers of the Association shall be a President, two Vice-Presidents, a Secretary and Treasurer, the latter two offices being vested in one; also three specially chosen active members to be known as Councilors, who, together with the officers, shall constitute the Council of the Association. The said Councilors shall be elected as follows: one for one year, one for two years, and one for three years.

Article 5. There shall be a Committee on Admissions and Ethics. It shall consist of the President and the Secretary and three other members, not members of the Council. These members shall be elected at the annual meeting at which this Amendment shall be adopted for the following terms: one for three years, one for two years and one for one year. At each subsequent annual meeting one member shall be elected for the term of three years to fill the annual vacancy. No member of the Committee shall be eligible for re-election until after one year shall have elapsed from the end of his term of service. It shall be the duties of the Committee on Admissions and Ethics to examine the merits of a candidate, to investigate any charges made against a member, and to report the results of the examinations and investigations to the Council in writing in the shortest possible time. It shall also be the duty of this committee to study the active work in science and practice of gastro-enterology accomplished in this country and Canada and to invite the meritorious workers to join this Association.

Article 6. The election of officers, Councilors and of the members of the Committee on Admissions and Ethics shall take place at the annual meeting and shall be by ballot.

Article 7. Vacancies in the offices occurring in the interval between the annual sessions shall be filled temporarily by the Council.

Article 8. The annual meeting of the Association shall be held at a time and place to be decided by the Council.

Article 9. The President shall be the chairman of the Council.

Article 10. There shall be annually two meetings of the Council; one shall take place at the call of the President, shortly after the annual meeting of the Association; the other shall take place at least four weeks previous to the annual meeting. It shall be within the discretion of the President to conduct this latter meeting by correspondence.

Article 11. The Council shall manage the affairs of the Association in accordance with the Constitution and By-Laws, and its minutes shall be reported to the Association at the annual meeting.

Article 12. This Constitution may be amended by a two-thirds vote of all the active members at any annual meeting, provided that notice of proposed amendments has been given in writing to all the active members of the Association by the Secretary, and provided further that such proposed amendments shall have been submitted to the Council and held over for one year.

Article 13. Any member failing to attend three consecutive annual meetings without an excuse acceptable to the Council shall be dropped from the roll. The Secretary, however, shall be required to call the attention of a delinquent member to the facts in his case previous to the third meeting.

Article 14. This Association adopts the Code of Ethics of the American Medical Association. Charges of advertising, of publishing knowingly false scientific statements, of actions unbecoming a gentleman and a high standing physician, etc., shall be investigated by the Committee on Admissions and Ethics. The accused shall have liberal opportunities to clear himself of the accusation. The Committee shall report its findings to the Council, and on the recommendation of this body an offending member may be expelled by a three-fourths vote of those present at the annual meeting.

BY-LAWS.

Article 1. The President and Vice-Presidents shall discharge such duties as are implied by their respective offices. The President shall preside at all sessions of both Council and Association.

Article 2. The Secretary shall attend to the usual clerical duties of the Council and Association. As Treasurer, he shall keep a record of the payments and arrearages of dues, and report on both at the annual meeting.

Article 3. The Council shall report its transactions to the annual meeting of the Association. It shall superintend the publication of scientific papers, but shall not appropriate for such printing an amount exceeding the sum of $100, without the vote of the Association. The order of business shall be arranged by the Council before the annual meeting. It shall pass upon the eligibility of candidates for membership.

Article 4. The yearly dues of active members shall be five dollars, payable in advance. Any member whose subscriptions shall be in arrears more than two years shall be reminded of the fact by the Treasurer, in writing in event that payment be not then made he may, on vote of the Council, be dropped from the roll of the Association. It shall be the duty of the Treasurer to report all members who are in arrears to the Council.

Article 5. When sufficient money is in the treasury the membership dues may be omitted when decided by the Council.

Article 6. These By-Laws may be amended, repealed or suspended by a two-thirds vote of the members present at any meeting of the Association.

ARTICLE 7. The quorum of the Association for the transaction of business, but not for the reading of papers, shall consist of seven active members.

ARTICLE 8. All titles of papers submitted for reading at the annual meeting shall be sent to the Secretary not later than four weeks before the date of the meeting.

ARTICLE 9. A typewritten abstract of not over three hundred words, of every paper to be read, must be sent to the Secretary at least four weeks before the annual meeting. It shall be the duty of the Secretary to send to each member of the Association a complete list of the papers to be read at the annual meeting, with their full titles, at least two weeks before the annual meeting.

ARTICLE 10. Papers submitted to be read before the Association shall not have been previously published or read elsewhere. Papers shall not exceed the limit of fifteen minutes without the consent of the majority of members attending.

PRESIDENT'S ADDRESS.

Delivered by the Acting President,

DR. FRANKLIN B. WHITE,

BOSTON, MASS.

THE ASSOCIATION, ITS AIMS AND MEMBERSHIP.

We all regret most heartily the absence of our dear President, Dr. Brown, and I am sure you will all join with me in every good wish for his welfare and speedy return.

He is one of the men in the Association with an active mind. He has plenty of original ideas and would have given us a great meeting.

This is not a formal presidential address, but I shall take this opportunity which I may never have again, of speaking informally of some things which the members ought to know and which I believe will greatly help our Association. As former Secretary I had a great opportunity to see what was going on, especially when the Secretary was also Treasurer and Recorder. I have seen many things which the average member has not learned or has forgotten.

I have watched the development of a compact group of men, which now includes the majority of the medical consultants throughout the country who are especially interested in digestion, also a number of skillful abdominal surgeons, several experienced roentgenologists, two physiologists, one or two pathologists and one or two physiological chemists. We have had no resignation for several years, we have a comfortable waiting list, and our meetings are larger and better attended and grow in interest.

There seems to be a real place for this Association. The development of a new section of the American Medical Association should make no real difference to us, there ought to be a general forum where all may go. We must expect to be a compact group of experts with a membership reasonably small where men from different parts of the country may get to really know each other, and where the discussion is of a more intimate sort. In other words, we must fill our place like any other national society.

Our active membership has grown from about 40 to over 80 in the last ten or twelve years. The ultimate size of our membership is open to question. We can afford to go a bit slowly and carefully select our material.

We do not want a man simply because we know him, or because he is a fine fellow; or even because he is a leading clinician or surgeon; but because, according to our constitution, "he has published meritorious work in gastro-enterology" research work, clinical studies, etc. This essential is sometimes forgotten in proposing men for membership.

It does not seem too much to ask that our candidates in the future should have a wide-spread reputation derived from their work and published papers in our field. We want to be sure that a man will help the Association not simply that the Association will help him. We want the very best men we can get throughout the country. All sections should be represented, including the far west, even if the men cannot always attend our meetings. We have one member from California, and another is on our waiting list.

The members have a real responsibility in proposing a candidate, whether scientist, clinician or young worker. They should write the Admissions Committee and tell them why he will help the Association and give all the information possible. Men are sometimes proposed carelessly or out of good nature or friendship and without letters and our essential qualification is forgotten.

Bastedo's plan, which was approved last year, for safeguarding our membership, is a valuable one. The personnel of the Association is very important, and we want to be fully informed about each candidate. Our previous methods of selection of candidates do not give opportunity enough for a free expression of opinion about each applicant.

In the first place we want a formal paper for each candidate giving the degrees, teaching position, society membership and publications in chronological order with a complete list of references or reprints.

In the second place, we want all this information (not simply the candidate's name) in the members' hands at least a month before our annual meeting. Some societies make it six months before the meeting, which is a good rule in some ways. We are in no hurry about candidates now and can afford to take our time and look them over well.

Another point: The long life of any association depends on the constant addition of the best young workers. "Young men for war, old men for council." If we are going to make successful war on the diseases of digestion, we must have the young men with us. We have no lack of able clinicians of wide experience to discuss and help us to digest and absorb or reject new work, but we must always have new material to live on.

We have members in practically all the large clinics, in all the large cities throughout the country, such as St. Louis, New York, Cleveland, Philadelphia, Baltimore, Boston, Rochester and many others to keep on the watch for clever young workers, not only to invite to our meetings and enrich our program, but to add to our Association.

No well-known or brilliant worker in our field, whether young or old, should be left outside the Association. They should be urged to apply for membership, or if sufficiently prominent, invited to join by the Admissions Committee. The members could help the Admissions Committee by pointing out good new members.

On the other hand, our own members must not rest on past laurels. We might do as some other societies have done, and create an emeritus class which shall include those members whose activities are slowing down and who have not made any real contribution to medicine for several years.

We may well consider the question of enforcing attendance at our meetings as a requisite for active membership. Do you call a man an active member who has not attended a meeting for four or five years in succession? He may be called a sustaining member, perhaps, but certainly not an active member. Perhaps such men who rarely attend our meetings and who do not wish to resign, may also be promoted to the emeritus class.

Our program: We must make our annual meeting the natural place where most of the best work of the year in gastro-enterology will be read and discussed. We must play the broad part of a national society in the best sense of the word. Our program must include not only the best work of our members but also the best work of the best workers they know. There is room for more real team work by the Association as a whole on our annual program. At present the President and Secretary do most of the work of getting the program together. I think it would be better if you all helped more. The meeting is your meeting, not the President's meeting. The members make the meeting possible by contributing valuable papers and discussions. They could also help by giving the officers their suggestions about the program, symposia, men to be invited, exhibits and social features, such as the banquet or smoker. I hope you will all take this seriously another year.

In this way we can make our Transactions a year book of the very best work in the country in digestive diseases, the best research, the best experimental work, the best clinical studies, the best statistical work and the best discussion. This is one of our important functions. There is no other association which is so appropriate a place for such reports.

It is interesting to note that in the program of the Association of American Physicians which meets here this year and which includes many of the finest medical men in this country, out of a list of 50 papers there are only three on the digestive tract, two of which are contributed by our own members; we are drawing the best work and the best workers in gastro-enterology into our own Association. In fact, there are only two places in the country where such a program as the present one can be gotten together, one at the section on gastro-enterology of the American Medical Association, the other at our own meetings. This is as it should be.

We have an opportunity to help the Recorder in the immense amount of labor which he silently performs and which no one can appreciate unless he, too, has been a Recorder. There is often a real difficulty in securing papers after they have been read and an extraordinary and utterly unreasonable delay in correcting discussions, which ought to be abolished, and will be if we all recognize it and do our part. I know all the excuses, I have used some myself, but they are not good excuses. We could easily make the rule that papers not sent to the Secretary or Recorder within a month or two after the session, say before July 1st, will not be published in the Transactions. The papers will naturally be fully prepared before the session and, if not handed in then, the final polish need not take over a week or two after returning home. The Transactions have steadily grown in size and the work of the Recorder has become so great that it is not fair that he should have to pursue the members indefinitely for the simple purpose of securing their papers and discussions for publication. The members can, and I am sure will show their real interest in the Association to the extent of giving this matter the preference over other extra work in the month following the session. Actual illness of the reader is the only satisfactory excuse. I feel sure that the excellent business management of our new Secretary will ultimately bring about the happy result that all the papers which are read will be handed in on the day of the session.

Our Association has made great headway and with a little more team work and devotion in the matter of membership and meetings, we can easily make our Association stand for the very best there is in the country in Diseases of the Digestive Organs.

APRIL 16, 1920.

DR. FRANKLIN R. WHITE,
322 Marlborough Street,
Boston, Mass., U. S. A.

MY DEAR DR. WHITE:

Your letter has just reached me, after nearly three weeks in transit from Boston, such is the irregular transportation by sea and land in these days of post-war reconstruction and economic unrest, so that in order that my answer may have any chance of reaching you in time for the meeting, I must write at once and can have time for only a few words although I should have loved to have sent a real paper, or at least a message worthy of you and of the Society.

It is needless to tell you and the members of the Association how deeply I regret not being at the meeting this year, not being able to renew the delightful personal intercourse which makes our annual meeting so stimulating, to listen to the papers which are always so interesting and so instructive, nor to draw again a long draught from the fountain of inspiration to help through another year of work, of teaching, of research along our chosen lines.

Perhaps one of the compensations of being *hors de combat*, and, as it were, living on a mountain afar off, is that it seems in a way to widen our vision, broaden our views so that, although the details must seem a little more blurred, the structure itself seems much clearer, both as regards itself and in relation to other lines of work and thought.

I have always felt, ever since devoting myself to this most interesting of the special subdivisions of the almost limitless medical field, that it had not yet received quite the recognition nor the appreciation that its importance warranted and I have tried many times to see how this could best be remedied, for no one recognizes the dignity, the importance, nor the possibilities of this field better than I.

My answer has always been that such appreciation and recognition can only come when our splendid Society as a whole and its individual members recognize to the full the marvelous potentialities in this our chosen field, as well as its inextricably intimate relationship to the whole field of general medicine and by precept and by word, by deed and by example, teach the real truth about gastro-enterology to the whole body politic of physicians.

Gastro-enterology can never be a narrow specialty; its fibres are too intimately interwoven in the structure of general medicine and with many of its more obvious subdivisions. Upon the physiologist, the internist, the radiologist, the chemist, the neurologist and the surgeon we are continually drawing for help and for information and to each one in turn we, too, are furnishing invaluable aid in their chosen field.

May my message simply be for one and all to preach and to practice the broad catholicity, not the narrow exclusiveness of our chosen field of work, to plan our future upon broad lines of inter-relationship rather than on the narrow basis of intensive specialization, for the older I grow and perhaps wiser become, the more I realize that the only large and wise and safe worker in our field must be he who has been and still is physiologist and physiological chemist, anatomist and radiologist, internist, and even albeit in a lesser degree, surgeon and gynecologist as well.

Will you express again to the members of the Association how deeply I appreciate the honor they have conferred upon me in making me their President for the past year, how deep has been my disappointment that I could not be with them this year and will you wish the Society and each of its members much happiness, much success and Godspeed for the ensuing year?

With kind regards and best wishes, I am,

Always sincerely yours,

THOMAS R. BROWN.

DIAPHRAGMATIC HERNIA DIAGNOSED DURING LIFE.

By Milton M. Portis, M. D.,

and

Sidney A. Portis, M. D.,

Chicago, Ill.

Diaphragmatic hernia is not so rare a condition as is commonly believed. Most of the cases in the literature were discovered either in the necropsy room or by operation. The diagnosis during life without operation was exceedingly rare until the advent of the Roentgen ray. With the aid of the fluoroscope in the last decade, cases have been reported frequently. Almost all of the roentgenologists of broad experience have reported one or more cases. The condition is encountered more frequently now as a result of war injuries.

Hernia of the diaphragm, as hernia elsewhere, may be of congenital or acquired origin. A distinction should be made between true and false hernia. The true hernia has a distinct sac, consisting of a layer of peritoneum inside and a pleural layer outside. Most of the cases of traumatic hernia, due to crushing injuries or stab or gunshot wounds, are of the false variety. The hernia may occur through the three larger preformed natural openings of the diaphragm which permit the esophagus, aorta and vena cava to pass, or the smaller ones for the passage of the azygos veins and splanchnic nerves. Besides, it may occur in congenitally weak areas of the diaphragm, like the foramen of Morgagni, which lies on each side at the costosternal portion, or it may occur in the posterior portion, in the space between the costal and lumbar portions of the diaphragm. Gossnitz* has shown by studies in comparative anatomy that this area is the last to be closed, and that defects invariably occur in this region.

Various abdominal viscera are found in the hernia, but the stomach and colon are by far the most common. The left side is the one most often involved, probably because the liver protects the diaphragm on the right side. According to Vogel,* the left side is involved 15 times as often as the right. The size is variable, from some so small that they can hardly be demonstrated, to others so large that almost the entire diaphragm is involved.

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*Gossnitz: Inaug. diss., Jena, 1903.

DIFFERENTIAL DIAGNOSIS ACCORDING TO ROENTGENOLOGIC FINDINGS.

EVENTRATION.	HERNIA.
High dome of the diaphragm with no loss of contour.	High diaphragm with loss of contour, a portion being regular, a portion being blurred.
No gas shadows above liver shadow.	Gas or barium shadow seen above liver shadow.
More likely to be congenital.	Gastric deformity.
Paradoxical movement of the diaphragm.	Obstruction.
	Presence of gas or fluid or both in the hernial sac.

SYMPTOMS.

There are no pathognomonic symptoms. Many cases give no symptoms at all. Especially is this true in those of congenital origin. The complaints are variable and arise either in the abdomen or in the chest, depending on the amount of interference with the function of the various organs involved in or near the hernia. The displacement of the thoracic organs by the hernia and the constriction of the hernial ring on the contents of the sac, as well as the effect of adhesions both inside and outside the sac, all serve to modify the clinical picture.

In some cases embarrassed respiratory action causes dyspnea and cyanosis. In others, cardiac displacement may cause palpitation and mediastinal discomfort and dizziness. Gurgling and rumbling sounds may be heard in the affected region.

Gastro-intestinal symptoms are not constant. Dyspepsia, belching, nausea and even vomiting may occur. Hematemesis has also been reported. Pain in the upper abdomen is frequent, and it may be felt in the chest on the affected side. The pain is usually increased by respiration. At times there may be colicky pains, with the clinical picture of subacute or acute intestinal obstruction.

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*Vogel, K. M.: Am. J. M. Sc., 145: 206-219, 1913.

Constipation is frequent, and strong cathartics may be necessary for free evacuation. Our case presents the clinical picture found in Hirschsprung's disease. The toxic picture, during the constipated stage with the accumulation of feces in the colon, was relieved by forced evacuations from time to time of large amounts of fecal material.

There is usually a loss of weight during the acute stage, with a gain in the interval, when free from symptoms. The position of the patient also influences the symptoms. Some are unable to lie down because of aggravation of the complaint, while others are able to lie only on one side.

Physical Signs.

In the large hernias the findings are usually definite and easily recognized. In the small ones, however, it may not be possible to demonstrate any abnormal findings.

The affected side may appear larger. Litten's sign is often absent on the affected side. The heart may be found to be displaced.

Tympany may be elicited where normal pulmonary resonance should be found. The note may change with posture or at different times, owing to the presence of fluid in the viscera in the hernia or fluid in the sac. This variation in the findings is very significant for the diagnosis of hernia. Breath sounds are usually absent over the tympanitic or dull area. Frequently gurgling sounds are heard. Succussion may be elicited.

The Roentgen ray findings are the most important and conclusive. With the fluoroscope one can readily see the high position of the diaphragm on one side, with one or more of the abdominal viscera protruding into the elevated area. With the opaque meal the stomach or colon or both may be demonstrated in the hernia. The displacement of the heart and the lungs and the change of position of the abdominal viscera are all readily seen. The stomach alone, or the stomach and colon, are found most frequently in the sac. The small intestine and the liver and the spleen have also been found in the hernia.

Differential Diagnosis.

Hernia of the diaphragm must not be confused with eventration, pneumothorax, subphrenic abscess or diverticulum of the esophagus. The differential diagnosis is usually not difficult. However, eventration offers a great deal of trouble in differentiating it from large hernias. The term eventration is rather loosely used by some authors. It should be limited to cases with atony or paralysis of half of the diaphragm. J. P. and D. Y. Keith state their differential diagnosis based on roentgenologic findings as in the accompanying tabulation.

In cases with hernia, bismuth can often be demonstrated in the colon above the level of the bow line of the diaphragm.

TREATMENT.

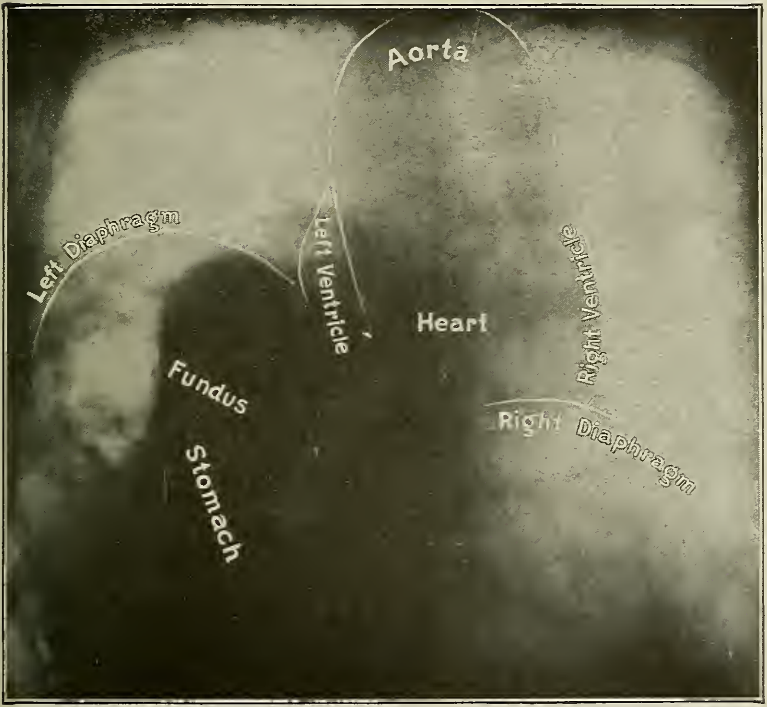
Aside from palliative medical measures, the only treatment is surgical repair of the hernia. Although the operation is at times very difficult, yet a number of successful operations are recorded in the literature.

REPORT OF CASE.

History.—Mr. M., aged 42, who consulted us in November, 1919, sixteen years before, while wrestling, felt a sudden severe pain in the left side, and this continued for several days. Since that time he had not been well. He had had recurring periods of abdominal distress, especially associated with constipation. By forced measures, a large evacuation was induced at these times and was accompanied by temporary prostration. Then he would have an interval of comparative comfort. These attacks had been coming more frequently until there were more than one a week. Between attacks stools were frequently ribbon-like and contained mucus, but no blood was ever noted. He had frequent headaches and malaise. There were attacks of severe epigastric pain in the early morning hours, which were relieved by alkalies, and he also had had sharp pains several hours after meals, which were relieved by food. He had never had nausea or vomiting.

Physical Examination.—The patient was well nourished and robust. The eye reactions were normal; the reflexes were intact, and likewise no abnormalities were to be found in the nervous system. The lymphatic system showed nothing abnormal. There was no deformity of the chest. The apex beat was seen faintly toward the ensiform. Litten's sign was absent on the left side. There was an area of tympany below the angle of the scapula on the left, and the breath sounds were faint in this region. Gurgling sounds were heard in the axilla and beneath the angle of the left scapula. The right border of the heart was found one inch to the right of the right sternal border, and the apex was displaced inward and downward. The heart tones were normal. The systolic blood pressure was 112, and the diastolic 85. The abdominal findings were negative. The liver was normal and the spleen could not be palpated. The urine showed no albumin, sugar or casts, but did show a large amount of indican. The stomach test revealed normal acids and ferments, and there was no occult blood. The motility was normal. The stool showed no occult blood. The blood gave a picture of secondary anemia of moderate grade. The Wassermann test was negative. The fluoroscopic examination revealed a high diaphragm on the left side which involved the inner two-thirds. In this pouch gas-filled viscera were seen. The heart was crowded to the right.

On giving barium in buttermilk, the stomach was found in this pouch and to its left the splenic flexure of the colon was seen. This was more apparent when the patient was in the reclining position. The stomach did not show an hour-glass appearance. Food dropped readily from the part above the bow line of the diaphragm into the part of the stomach below and the stomach emptied in normal manner. The duodenal bulb was normal. The liver shadow was normal and there was no tenderness over the appendix or the gall-bladder.



Left leaf of diaphragm high and arched; stomach in pouch; to right and below, splenic flexure of colon.

DISCUSSION.

Dr. White: The discussion on the paper of Dr. Milton M. Portis, of Chicago, entitled "Diaphragmatic Hernia Diagnosed During Life," will be opened by Dr. Seymour Basch, of New York City.

Dr. Seymour Basch, New York City: Dr. Portis has correctly stated that the diagnosis of diaphragmatic hernia has advanced very considerably in the last decade, mainly through the aid of the X-ray; and probably, too, from the fact that increasing knowledge has directed our attention to its occurrence and more cases have been discovered, and not because the condition is of greater frequency than formerly. Previous to the use of the X-ray, the diagnosis had sometimes been made during life by clinicians and, of course, it was considered a condition of great rarity.

The condition of diaphragmatic hernia is very easy to diagnose in our modern times, as hinted by Dr. Portis, when we have a pronounced, an extreme case; but, when we have a small hernia, such as might be produced by a stab or a pistol wound, or by spontaneous rupture of the diaphragm, the difficulty is much greater, and can be made only through repeated and careful examination. Such a case was reported by Agenthorn in which a loop of the small intestine was eventrated into the thoracic cavity. The X-ray showed the malposition, but did not show the hernia.

The two points that are important to remember regarding X-rays are first that whereas you see the upper line of the stomach, it is not, as Dr. Portis pointed out, the line of the diaphragm. The line of the diaphragm in a true hernia is broken and you can get the contrast with the other side by comparison. The line of the stomach is a large, pronouncedly white line and can be diagnosed readily, even without the stomach being filled. I will pass around the photograph of such a case, in which you can see the white line of the stomach and the broken line of the diaphragm.

A second point that is of importance in the differentiation between either the relaxation of the diaphragm in congenital conditions and true hernia is that in watching the fluoroscope and having the patient breathe deeply, the abdominal organs, in true hernia, usually move upward, whereas in congenital relaxation the respiration pushes them down. From the X-ray viewpoint, this is more important through the fluoroscope than through the X-ray plate, because with the fluoroscope you can, by changing the position, notice the line of fluid and see the organs almost as well as with the plate.

Among the physical signs, there are two or three that are of importance, one of which was not mentioned by Dr. Portis. That one is the displacement of the heart. Almost all of these hernias are on the left side; and as a result, the stomach being prolapsed upwards, the heart is pushed to the right. Still in the non-barium-filled stomach, with fluoroscope and upon the plate also, you can see the dark shadow of the heart somewhat to the right of and showing through the stomach.

As regards treatment, there is no doubt about the thoracic method being better for the repair of the hernia; but it has one drawback, and that is that you cannot inspect the abdomen and see whether there is any injury of its organs. You often find these and also adhesions to the diaphragm which cannot be seen by the thoracic route. Many operators use both routes: the thoracic first, and later the abdominal.

It is surprising how large a number of good results have accrued in the last six years from operative procedure, as compared with the lamentable results before. I want to pass these pictures around. One shows a real diaphragmatic hernia of the left side. The other is the case reported here six years ago, in which a hernia had occurred on the left side, close behind the esophageal opening, and had passed around and to the right side, to the level of the second rib. It shows the position of the stomach beautifully.

Dr. Franklin W. White, Boston: If the Association will permit, I shall be glad to show my lantern slides of the case on the program, entitled "A Brief Report of an Unusual Case of Elevation of the Left Diaphragm (Eventration), with Inverted Stomach," as a part of the discussion on the paper of Dr. Portis.

Now a word about the diagnosis. The real important thing is to distinguish it from a high-sided left diaphragm, which I think is a better term than eventration. The high-sided diaphragm is not a surgical condition, eventration is. Physical signs do not distinguish the two things. We get the splashing associated with air in the lower part of the chest in both conditions, and can make the clear diagnosis only by X-ray examination. Some things have been put down as diagnostic that are not of great value in my opinion. Respiratory movement on both sides does not help in distinguishing high-sided left diaphragm from hernia. The hernia may move up and down and the diaphragm may be immobile. The movements must be equal with respiration and must be in the normal direction. The dome-shaped line is considered characteristic of the left-sided high diaphragm. If we have the right kind of hernia, we get a symmetrical dome-shaped line, even in hernia. The matter of so-called paradoxical respiration, the right side going down and the left going up, is not a good sign, because it is too rare to be a good diagnostic sign.

So we come down to two or three things. Picking out those conditions, one is the great variability at different times that we get with hernia, and the great irregularity at different times—half way up and half way down, etc.—in hernia. We also see the muddled (?) line in many a hernia, and not in the high-sided left diaphragm. Furthermore, we almost always get this doubled-up stomach in hernia; while in high-sided diaphragm the stomach is likely to hang.

Dr. Charles G. Stockton, Buffalo: There are one or two points that I should like to speak of. The first is the difficulty in diagnosis. It is 30 years since I had my first case of diaphragmatic hernia; it seemed impossible to make any other diagnosis—of course, on clinical grounds. The case was so perfectly definite, with the ascent of the abdominal viscera into the thoracic cavity, taken with the displacement of the heart and the striking obstruction in the digestive tract, and also the obstruction to circulation and respiration, that there was no other diagnosis possible. There were marked cyanosis, great dyspnea, vain attempts at vomiting and a comparatively motionless left chest. There were tympany, musical sounds and tinkling; they were the sounds of the intestinal movements, borborygmi, and could be clearly made out in the chest. The left chest was overdistended. I had never heard of diaphragmatic hernia but made the diagnosis because I could not make any other. Minter, of Copenhagen, then resident in Buffalo, laughed at me for making it, and said that he had never heard of such a thing. The diagnosis was maintained by a comparative boy in the profession, and yet not found by an expert. The autopsy showed that the boy was right. I felt, from then on, that it was comparatively easy to make a diagnosis of diaphragmatic hernia. I taught that, when I became a teacher; but I found that I did not fully appreciate the difficulties. A later case that I diagnosed from clinical examination as hernia, the X-rays indicated to be eventration. I am yet in doubt about the diagnosis. I wish to point out that it is not always easy, even by means of the X-ray, to differentiate between these conditions. Finally, I had to confess that a case that I had said was something else was proved at autopsy to have been one of diaphragmatic hernia. Sometimes the diagnosis is perfectly obvious. The question of obstruction to circulation and breathing should be taken into consideration.

Dr. Joseph Sailer, Philadelphia: I cannot agree with Dr. Portis that eventration of the diaphragm is a condition due to paralysis of one-half of the diaphragm, because in cases that I have seen the diaphragm moved freely. In one case that ended fatally, the muscle fibres were found in half of the raised diaphragm as well as in the other half.

Neither can I agree with Dr. White that we have no signs that enable us to make the diagnosis by means other than the X-ray.

I agree with Dr. Smithies that the X-ray may be misleading. The differences are two. In one, we have a symmetrical chest with symmetrical expansion. This has been noted in all congenital cases.

With regard to eventration, this is not a disease of the diaphragm but of the lung. The diaphragm merely rises to allow the abdominal contents to take the place of the hypoplastic lung on that side. In diaphragmatic hernia, on the other hand, we have marked difference in the expansion of the chest. It is characteristic, but not, of course, characteristic of diaphragmatic hernia in any way, because there are other conditions in which there is limitation of movement of one-half of the chest. The best distinction, I believe, is von Leyden's sign. Von Leyden, in writing his masterly paper on "Subdiaphragmatic Pneumothorax," called attention to it. It consists in the fact that when the area of tympany is percussed, it is found when the patient takes a deep inspiration that the area of tympany descends in the congenital form of eventration; whereas in diaphragmatic hernia, the area of tympany does not move. In one case of diaphragmatic that I saw, it did not descend, but as Dr. White says, it showed a variability, depending on the degree of hernia. The diaphragmatic movement in eventration is added proof that we have to do with a displaced diaphragm that is distinctly muscular. It is possible that the diagnosis of eventration may also be suspected by tracings with the electrocardiograph, which may indicate the displacement of the heart. This is merely a suggestion, for I am not aware that it has been tried.

Dr. Willy Meyer, New York City: The development of work in the last year, it seems to me, has brought a great advantage in the clear and more definite diagnosis of these conditions. I am referring to the so-called pneumo-peritoneum, the injection of oxygen into the peritoneal cavity. Those who have seen the X-ray or lantern slides of this method will realize that it should be used only on strictest indication; they will also agree that the line of the diaphragm is shown most beautifully. Whether it is advisable to employ the method in cases of diaphragmatic hernia, extended experience alone can prove. Two years ago, at the Cincinnati meeting of the American Surgical Association, the case of a boy was related in whom a simple treatment after the bismuth meal had been made use of. The radiographist stood the boy on his head, and now the bismuth gradually entered that part of the stomach above the diaphragm and pushed out the air bubbles. The result was most convincing.

With regard to operative treatment, it was mentioned by Dr. Basch that the combined operation is the one that should be used. If I had to operate on such a case today, I would first do abdominal section, and then at once add a transpleural incision into the chest. By this means we can not only push down the intestinal contents that have wandered into the thoracic cavity after thorough separation, but also pull them down at the same time with the hand within the abdomen. We can then also look into the peritoneal cavity and see that everything is in normal position as far as possible.

Dr. Lewis Gregory Cole, New York City: I want to mention one case which might have been included in Dr. Portis' series. It was the first of such cases that I have seen and was the fourth case on which I did any gastro-intestinal work, so it must be one of the early cases reported. It was reported by Dr. Emmett Holt and might have been 15 years ago, and I do not recall just where it was published.

It was a very characteristic case in a child and undoubtedly can be found.

The only other thing that I want to speak of is that I want to warn you against the premature diagnosis of these conditions on such evidence as has been submitted. It may seem strange to you for me to utter such a warning, but I do wish to utter one against differential diagnosis of these conditions on such evidence as has been submitted here today. I realize that the plates shown may be old and done at a time when the present perfection of X-ray diagnosis did not exist. This comment is not intended as disrespectful to anyone, but I do want to warn you that in the future you should have more definite evidence.

Dr. R. Walter Mills, St. Louis, Mo.: Dr. Basch had in his collection plates of diaphragmatic hernia that he did not show. These bring up several points, that Dr. Cole accentuated. One print shows what may be termed pseudo-diaphragmatic hernia. One sees what might be interpreted as evidence of diaphragmatic hernia; an apparently deformed stomach with gas bubble of such shape and high position as to strongly suggest diaphragmatic hernia. The condition, however, is not due to diaphragmatic hernia but a peculiarity of body statics usual in hypersthenic subjects, namely a marked antero-posterior body depth with resulting spacious upper abdominal cavity, a condition often accentuated by an emphysematous thorax. In such an abdomen of great depth the stomach lies largely antero-posteriorly, is very high, often straddles the transverse colon and, as viewed from in front, frequently presents curious outlines simulating organic defects. These artefacts are not only mistaken for diaphragmatic hernia but are sometimes operated on for hour-glass stomach or cancer. Their misunderstanding has helped to give a black eye to X-ray interpretation. There is another condition that simulates diaphragmatic hernia. There are cases in which the stomach gas bubble is apparently above the level of the convexity of the diaphragm. This is so because the diaphragm is not necessarily an even convexity. The line we speak of as the line of the diaphragm is due to the fact that at its maximum convexity, the diaphragm being there approximately in a transverse plane, locally offers greater resistance to the rays passing in an antero-posterior direction; it is hence projected on the plate as an arc like outline that we speak of as the diaphragm. If the diaphragm be not of even convexity as sometimes occurs in the same hypersthenic habitus, there occurs a second higher bulging posteriorly into which the fundus of the stomach is elevated. The gas bubble of the stomach thus appears above the diaphragm, yet it does not represent a diaphragmatic hernia. I have seen such a case operated on for diaphragmatic hernia. It accentuates that the X-ray findings should be interpreted after broad experience and with the utmost conservatism.

Dr. Portis (closing): I wish to thank the gentlemen for the discussion of the cases submitted, of which I should like to have the report for the final paper. I have given just a brief resume of what appears in the paper. I am glad to get the warning concerning the difficulty of differential diagnosis. In the various reports of cases, I found that some of the hernias were so small that they could hardly be diagnosed at operation. One should look very carefully all through the dome of the diaphragm, to see whether a little hernia has not occurred, particularly in the gastro-lumbar portion. Most of those not of congenital origin occur in this congenitally weak portion, and some have recently occurred as the result of war injuries. We shall probably find more of these in the next few years.

DIAGNOSIS AND TREATMENT OF RECURRENT CHOLECYSTITIS WITHOUT STONES.

By Max Einhorn, M. D.,

and

Willy Meyer, M. D.,

New York.

The last two or three years have given us frequent opportunity to deal with gall-bladder inflammations without stones in which the diagnosis as well as the treatment presented a rather difficult problem. Although some surgeons, notably the Mayos, Dr. R. S. Fowler,[1] of Brooklyn, and John B. Deaver,[2] of Philadelphia, excise the gall-bladder when chronically inflamed, the majority of clinicians and not a few surgeons doubt the advisability of extirpating a gall-bladder—even if diseased—so long as it does not contain stones. Inasmuch as our opinion is contrary to this commonly held view, we thought it best to present our experience regarding this subject before you.

As the basis of this paper we select 18 cases of recurrent cholecystitis without stones that have been operated upon, and in this way the diagnosis corroborated *per autopsiam in vivo.* A few of these cases we shall describe in detail while all will be mentioned in the table stating the findings of the duodenal contents.

Case I.—Albert De V., 25 years, October 22, 1919. In January, 1919, patient was seized with a severe, sharp, knife-like pain, lasting several hours. The attacks occurred at intervals of about once every month up until July, 1919. Since that time they came on every week to ten days. The pain had no relation to food, radiated to the left side and was relieved only by hypodermics of morphine. There was no vomiting, no belching of gas, bowels were normal. During the attacks the patient would lose two or three pounds, but he would regain this weight in the intervals of euphoria. He was jaundiced once shortly after an attack. Patient has always been well prior to the present illness.

Examination of the abdomen revealed a slightly enlarged liver. There was an area below the right costal margin painful to pressure. The gastric contents showed: HCl+; acidity=65; the string test revealed a blood stain at 18 inches' distance from the lips. The duodenal contents aspirated in the fasting condition were turbid, grayish green looking. A diagnosis of gastric ulcer and cholecystitis was made.

Patient was treated by duodenal alimentation and was then operated upon by Dr. Willy Meyer for his gall-bladder condition. Wassermann was negative.

November 21, 1919, operation: appendix tightly adherent to meso-appendix, old bands; typical removal. No stones in gall-bladder which is blue, glistening, soft. Aspiration; contents turbid; cholecystectomy; firm adhesions between liver and bladder; large adherent glands around cystic duct. Appendix contains large fecal concretion with proximal stricture. Culture from aspirated bile of gall-bladder showed colon bacilli.

March, 1920.—Patient feels perfectly well; has had no attacks since the operation.

Case II.—Mrs. Rose S., 38 years. February 4, 1920. The patient gave a history of abdominal cramps on and off for 18 months. Five weeks ago had severe abdominal cramps in right epigastric region radiating to right shoulder associated with some jaundice and light colored stools; two weeks later another attack came on, and since then has had them daily. No vomiting. The appendix was removed two years ago. Upon examination a mass is palpable in the region of the gall-bladder, which has an oval configuration of about one and one-half to two inches in length and one inch in width and feels elastic and somewhat pliable. This appears to be the gall-bladder. Temperature upon admission until operation, normal to 100°.

Duodenal contents: Greenish yellow; turbid; alk=20; A=+; S=+; T=+; microscopically, bilirubin calcium and cholesterin crystals; bacteria.

Blood count=R. B. C., 4,600,000; W. B. C., 8,800; polys, 80 per cent.; lym., 20 per cent.; Hb., 80 per cent. Wassermann negative; String test negative; X-ray negative.

Gastric contents: HCl, 0; Acidity, present; Blood+.

On March 8, 1920, cholecystectomy was performed by Dr. Willy Meyer. The bile aspirated from the gall-bladder at operation was dark brown and turbid.

Case III.—Mrs. Eva B., 43 years old. March 20, 1919. She has had attacks of pain in the epigastric region on and off for five years. Pains radiate across abdomen and are accompanied by vomiting. Sometimes they come on after meals and at other times they have no relation to food. Two months ago the patient ate some chopped meat and an attack came on. This brought her to the hospital. Jaundice was present two years ago.

Temperature normal up until the time of the operation. Duodenal contents: yellowish brown, turbid; alk=25; A, 6; S, 3; T=5. Gastric contents: HCl=20; Acidity=40; Blood, +.

Cholecystectomy and appendectomy performed by Dr. Willy Meyer on April 1, 1919. The gall-bladder was glistening, not blue; the bile aspirated from the gall-bladder at operation was greenish and very turbid. Pathological diagnosis: Chronic catarrhal cholecystitis; chronic appendicitis.

At the operation the following was found: Pyloric ring irregular, slightly infiltrated; pannus, with adhesions upward, but not to liver; probably additional duodenal ulcer, but clinically symptoms missing.

Later on at home, she had vomiting spells and was then readmitted to the hospital. No food retention was found in the stomach. She recovered.

Case IV.—John A. H., aged 29 years, admitted May 5, 1919. Four weeks ago patient became dizzy; felt like fainting; two weeks later had pain across the abdomen, but it did not radiate and had no relation to food. The bowels were constipated since onset; no vomiting.

Physical Signs:—Tenderness in right upper quadrant. The edge of a tumor mass can be made out in right upper quadrant. The temperature on admission was 103°, then varied from 102° to 105° until the operation, after which it fell to normal in ten days.

Duodenal contents: yellow, very turbid; alk=25; A=6; S=1; T=1; many bacteria, pus cells, and cholesterin and calcium bilirubin crystals. Blood count: W. B. C.=20,400; polys.=86 per cent.; lym., 14 per cent.

Cholesterol=120; bile sterile; Widal negative; complement fixation for tuberculosis negative; Wassermann—0; gastric contents, HCl.=50; acidity=75; feces negative.

On June 5, 1919, a cholecystectomy and appendectomy were performed by Dr. Rehling.

Pathological Diagnosis:—Chronic periappendicitis; catarrhal cholecystitis.

Case V.—Max S., age 50 years, admitted February 17, 1920. Five weeks ago the patient had a gnawing pain in midline region of the stomach, four hours after the noonday meal; the pain was relieved by taking food; no vomiting, patient belched gas after meals, lost 15 pounds in two months. Wassermann was positive two years ago; had ten salvarsan injections. The bowels were regular.

Physical Signs:—Liver palpable two fingers below the epigastric angle. Heart sounds of poor quality; short systolic murmur apical; pupils sluggish to light.

Duodenal contents: yellow pea-soup appearance; turbid; alk=20; A=+; S=2; T=6; cholesterin crystals; bacteria and mucus. Gastric contents: HCl.=14; acidity=26; no blood; string-test negative; feces negative. Wassermann negative at present. Blood count: W. B. C., 5,720; polys., 58 per cent., lym., 42 per cent.; Hb., 80 per cent. Temperature, slight elevation up to 100. The X-ray apparently showed penetrating gastric ulcer; persistent defect in the lesser curvature.

Cholecystectomy was performed by Dr. Willy Meyer on February 23. No ulcer was found. Bile aspirated from the gall-bladder, alk=120, dark green like Kulmbacher beer.

Case VI.—Jacob R., age 40 years. Admitted September 30, 1919, with a history of having suffered with pain in the right upper quadrant for four years, the attacks coming on gradually and leaving gradually, the pain radiating to both shoulders; no jaundice; lost 30 pounds in three or four months.

Physical examination shows slight rigidity and extreme tenderness over the entire right side; the liver is enlarged and very tender at the site of the gall-bladder. X-ray negative; Wassermann, 0; blood count, W. B. C.=7,550; polys.=62 per cent., lym.=38 per cent.; R. B. C. 6,000,000. Temperature, 102°.

Duodenal contents yellow with a tinge of green, turbid; alk=30; A=8; S=1; T=1; microscopically are seen cholesterin crystals, bacteria and white blood cells. Gastric contents: HCl= 66; acidity=82; blood negative. String test negative.

Cholecystectomy and appendectomy performed by Dr. Torek on November 1, 1919. The contents of the excised gall-bladder=culture sterile. Pathological diagnosis, catarrhal appendicitis; slight localized inflammation of the gall-bladder. The temperature dropped to normal two days after the operation.

CASE VII.—Mrs. D. Sick for many years. Frequent pain in the epigastric region and had to be most careful in diet; gained 20 pounds lately under proper management; this speaks much against carcinoma though the presence of acanthosis nigricans suggests the same. At the first office call tenderness was found in the region of the gall-bladder; duodenal contents found turbid with cholesterin crystals.

February 17, 1919. Operation; stomach large and ptosed; no sign of ulcer at pylorus or duodenum nor of carcinoma in entire length of stomach, head of pancreas, large intestine or liver; gall-bladder large, not blue glistening, evidently chronically inflamed; appendectomy. Aspiration of the gall-bladder shows pronouncedly turbid bile; cholecystectomy. Bacteriological examination of the aspirated bile from the excised gall-bladder showed it to be sterile.

In conjunction with the above cases we give in the following table a detailed description of the duodenal contents as found in all the patients before the operation, and in some also of the bile aspirated from the gall-bladder during the operation.

In looking over the cases with regard to their symptomatology, two types can be easily discerned. Type I is characterized by distinct attacks of colicky pains in the upper abdomen (usually on the right side or exceptionally on the left side), lasting from one to several days, followed by perfect relief of symptoms. One or more such attacks will have occurred before the patient consults his physician. During the attack there is often a slight rise in temperature accompanied by a moderate leucocytosis and an increase of the polymorphonuclear white blood cells.

Type II shows no distinct attacks. The patient usually complains of slight distress either in the mid-epigastrium, especially after meals, or somewhat to the right of the linea alba covering, principally the right hypochondriac region. While there is usually no rise in temperature, at times the latter may occur in a pronounced manner. The patient is then debilitated and bedridden.

In both types the objective findings are as follows:

The liver is frequently found somewhat enlarged. Palpation to the right of the linea alba in the area situated between the costal margin and the navel line occasionally elicits pain, especially during an inspiration. In the same region there is at times found the *défense musculaire* of the rectus abdominis muscle.

The bile, as found by aspiration of the duodenal contents in the fasting condition of the patient, is usually turbid, presenting a yellow-brownish appearance. Occasionally it looks greenish. Microscopically numerous bacteria and cocci are frequently found; often pus cells; cholesterin- and bilirubin-calcium crystals are likewise encountered, but in comparatively small numbers. The alkalinity and the pancreatic ferments do not, as a rule, deviate much from the normal.

In the above-mentioned 18 cases of true cholecystitis verified by operation and pathological examination, the duodenal contents were pretty clear in one (Mr. B., No. 11), while the rest showed pronounced turbidity with all the attributes mentioned in the previous paragraph.

In quite a number of these patients the appendix was found to be considerably diseased. In every one of the patients (excepting a few who had undergone appendectomy before) the appendix was removed as a prophylactic measure. A few of the cases were complicated with peptic ulcers, requiring special treatment for the latter affection.

At the operation the gall-bladder, as stated above, was thoroughly examined by inspection, palpation and aspiration. In all the cases mentioned it was found diseased—either thickened or glistening whitish, or the bile was dark black and turbid or containing much mucus and slightly thickened material. The pathological anatomical diagnosis, as given by the pathologist of the hospital, is stated in each case examined. The gall-bladder was always found diseased and the diagnosis of cholecystitis corroborated.

Diagnosis.—The recognition of chronic cholecystitis without stones is not difficult. In making a diagnosis, however, in most instances, merely the cholecystitis can be made out, while the absence of stones cannot be foretold until after the operation.

According to Smithies,[3] even in distinct cases of biliary colic, stones are found in 57 per cent., while in 43 per cent. no calculi are present. In exceptional cases the probable occurrence of a cholecystitis without stones can be predicted.

The diagnosis of cholecystitis is based upon the symptoms (colicky pains in the upper abdomen; distress in the epigastrium radiating backward to the right) in conjunction with the presence of pronounced turbid bile in the duodenum in the fasting condition of the patient, as found by means of the duodenal tube. The existence of a considerable leucocytosis, with an increase of the polynuclear elements, enhances the diagnosis and indicates the severity of the lesion.

Treatment.—Mild cases of cholecystitis yield often to medical treatment. The same consists in ample rest, frequent small meals, plenty of water, saline aperients (Carlsbad, Saratoga, French Lick Springs) washing of the duodenum with a weak argyrol or ichthyol solution (1 to 2 per cent.).

Frequently recurring attacks of cholecystitis, especially when accompanied by even a slight rise of temperature, require operative intervention. The same applies to forms of cholecystitis throwing the patient into a state of invalidism or endangering life by infection. The operation consists in removing the offending organ (cholecystectomy).

The Reasons for and the Results of the Operation.—The reasons for the operation in the cases described were: (1) Recurrent troubles not yielding to the usual modes of treatment. (2) Febrile conditions bearing the stamp of infection and apparently due to the gall-bladder, endangering life.

The results were uniformly good, excepting in one case in which recurring attacks of jaundice and cholangitis persisted after the operation.

Conclusions.—1. A diagnosis of chronic recurrent cholecystitis can frequently be made, even when stones are absent, by the symptoms present in conjunction with a direct examination of the bile.

2. When operating for cholecystitis a chronically diseased gall-bladder should be excised, even if no stones are found in the affected organ.

M. E.

TABLE OF EXAMINATION OF THE DUODENAL CONTENTS (BILE) IN 18 CASES OF CHRONIC CHOLECYSTITIS WITHOUT STONES OPERATED UPON.

Name.	Date.	Alkalinity.	Amylopsin.	Steapsin.	Trypsin.	Appearance and Microscopical Findings.	Remarks.
Mrs. M.	4-15-18	20	10	3	1	Yellow; slightly turbid.	Cholecystectomy by Dr. Willy Meyer.
Mrs. B.	3-30-19	25	6	3	5	Yellowish brown; turbid.	Appendectomy and Cholecystectomy by Dr. Willy Meyer; gall-bladder at operation glistening; not blue; bile greenish and turbid. Pathological diagnosis—chronic catarrhal cholecystitis; chronic appendicitis.
Mrs. D.	2-14-19	30	6	2	4	Very turbid; yellow; green, pea-soup appearance; cholesterin and calcium oxalate crystals.	Cholecystectomy by Dr. Willy Meyer.
Mr. A. de V.	11-2-19	25	11	2	5	Reddish yellow; turbid, pus cells; cholesterin crystals.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder turbid; culture showed colon bacilli; gall-bladder at operation glistening, blue, and soft.
Mr. K.	11-2-19	25	7	2	2	Yellowish green; turbid; pus corpuscles.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder greenish and turbid; at operation right lobe of liver infiltrated.
Mrs. G.	11-25-19	30	10	1	3	Yellowish green; turbid.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder dark green; turbid; pathological diagnosis—subacute catarrhal cholecystitis.

TABLE OF EXAMINATION OF THE DUODENAL CONTENTS (BILE) IN 18 CASES OF CHRONIC CHOLECYSTITIS WITHOUT STONES OPERATED UPON.

Name.	Date.	Alkalinity.	Amylopsin.	Steapsin.	Trypsin.	Appearance and Microscopical Findings.	Remarks.
Miss P.	2-6-20	20	10	1	2	Yellow turbid; small cholesterin crystals; calcium bilirubin crystals; pus cells.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder green and turbid.
Mr. R.	10-15-19	30	8	1	1	Yellow; tinge of green; turbid; pus cells; cholesterin crystals; bacteria.	Cholecystectomy and appendectomy, by Dr. Torek. Pathological diagnosis, catarrhal appendicitis; slight localized inflammation of gall-bladder.
Mr. H.	5-28-19	25	6	1	1	Yellow, very turbid; cholesterin crystals; bacteria; calcium bilirubin crystals; pus cells.	Cholecystectomy and appendectomy by Dr. Willy Meyer. Pathological diagnosis, chronic periappendicitis; chronic cholecystitis.
Rev. H.	1-25-19	15	5	Trace	0	Light yellow; turbid; few cholesterin and tyrosin crystals; mucus; pus cells.	Cholecystectomy by Dr. Willy Meyer. Pathological diagnosis, chronic cholecystitis.
Mr. B.	2-16-20	20	10	Trace	1	Golden yellow; clear.	Bile from gall-bladder at operation dark colored. Cholecystectomy by Dr. Willy Meyer. Pathological diagnosis, catarrhal cholecystitis.
Mr. S.	2-16-20	30	. .	2	6	Yellow; pea-soup appearance; turbid; mucus; cholesterin crystals; bacteria; pus cells.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder dark green like Kulmbacher beer.

TABLE OF EXAMINATION OF THE DUODENAL CONTENTS (BILE) IN 18 CASES OF CHRONIC CHOLECYSTITIS WITHOUT STONES OPERATED UPON.—CONTINUED.

Name.	Date.	Alkalinity.	Amylopsin.	Steapsin.	Trypsin.	Appearance and Microscopical Findings.	Remarks.
Mr. P.	2-29-20	20	. .	?	.	Grayish, reddish tinge; turbid.	Appendectomy and cholecystectomy by Dr. Willy Meyer; bile from gall-bladder dark and black at operation, gall-bladder shows enlarged vessels; bluish and glistening.
Mr. B.	3-21-20	20	6	1	5	Grayish; turbid; tinge of yellow; many cholesterin and calcium bilirubin crystals; mucus; ova.	Cholecystectomy by Dr. Willy Meyer.
Mr. D.	3-24-20	20	6	1	3	Greenish yellow fluid; mucus.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder dark, green; turbid; alk, 60.
Mr. F.	3-16-20	20	4	1	2	Yellow; turbid; cholesterin crystals; mucus; epithelial cells.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder turbid; dark green.
Mrs. S.	3-1-20	20	. .	.	.	Greenish yellow; turbid; small cholesterin crystals; calcium bilirubin crystals; bacteria; mucus; epithelial cells.	Cholecystectomy by Dr. Willy Meyer; bile from gall-bladder dark brown, turbid.
Mr. B.	4-20-20	35	8	1	3	Greenish yellow; turbid; cholesterin crystals; epithelial cells; mucus; pus cells.	Cholecystectomy by Dr. Willy Meyer.

ADDITIONAL REMARKS FROM THE SURGEON'S POINT OF VIEW.

In patients complaining of distress in the upper abdomen physicians and surgeons will think of a diseased stomach, duodenum, gall-bladder or pancreas; the appendix and kidney also come into consideration.

After we have obtained a most careful history of the case and made an equally thorough clinical examination, trying to make a preliminary diagnosis, the laboratory nowadays steps in for differentiation. Examination of stomach contents, string test, X-rays, Wassermann reaction, and stool analysis pave the way for the correct diagnosis of gastric or duodenal affections.

For the determination of gall-bladder disease there are at our disposal today, citing them in accordance with the time of their introduction: radiography, cholesterol test of the blood and examination of the duodenal contents.

The X-rays, according to some writers, show stones in about 15 to 20 per cent. of the cases; others have found positive results in somewhat over 30 per cent., while others still claim much higher figures. The rays cannot assist in shedding light on the condition of patients who suffer from an inflamed gall-bladder without stones, though they can show adhesions (insufflation of oxygen into the peritoneal cavity).*

The cholesterol test is generally believed to be unreliable.

The examination of the duodenal contents with the help of the Einhorn tube, chemically, microscopically and bacteriologically, bids fair to throw a great deal of additional light on the subject. At the present moment, it appears to be the best and most reliable means we possess for diagnosing an affection of gall-bladder or bile ducts.

For several years Dr. Einhorn and myself have systematically added the examination of the duodenal contents to the other laboratory tests, and then compared the condition of the bile as it is physiologically discharged into the duodenum previous to surgical interference, with later operative findings.

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*This at least had been the generally accepted opinion of clinicians up to date. The recent presentation of the subject before the American Gastro-Enterological Association, May 3, 1920, by Dr. A. W. George of Boston makes us entertain the hope that radiography is going to show disease of the gall-bladder in almost 90 per cent. of the cases. The rays frequently demonstrate a distinct contour line of the gall-bladder without stones. At the present moment it is believed that if they do so the gall-bladder is pathological. A healthy gall-bladder does not show on the plate under ordinary conditions. The pneumoperitoneum (oxygen insufflation), however, does do so.

We can state that the pre-, inter- and post-operative examinations tallied in almost every instance. It is, of course, necessary to leave the duodenal tube in place for a number of hours (3 or 4) and then aspirate every half hour. In some cases, *e. g.*, if the cystic duct is strictured or a stone completely obstructs it, the secretions will represent fresh liver bile only. If the examination before operation had shown the bile discharged through Vater's papilla blackish green or brown in color, and turbid, with cholesterin or bilirubin crystals and pus corpuscles, aspiration of the bile with a needle introduced into the gall-bladder during the operation usually showed the same thing.

Inasmuch as the culture of the bile aspirated during operation not infrequently failed to show bacterial growth, the question arose: Is a blackish-green or dark brown bile pathological? We believe it is. Physiologists teach that normal bile from the hepatic duct as well as from the gall-bladder is golden yellow in color. In animals possessing a gall-bladder, the bile within the latter has uniformly shown a golden-brown color. Furthermore, as stated by Dr. G. L. Rohdenburg, Pathologist of the Lenox Hill Hospital, "Two healthy young men in the twenties of life, who were autopsied within five minutes after death, the latter having followed legal electrocution, showed a golden-yellow bile in the gall-bladder." He adds: "The fact that death by this method raises the body temperature to 120°-150° F., may, of course, have had some effect on the fluid content of the gall-bladder." Again he states: "That some of the fluid from diseased gall-bladders is sterile, is not surprising; first, because bile is lytic for several types of bacteria; second, because it is probable that the bacteria do their damage in the tissues of the gall-bladder wall."

Personally I would add that possibly the frequent negative results of culturing bacteria in the dark green or brown turbid bile, as aspirated from the gall-bladder during operation, may depend upon the procedure itself. In order to give results the culture has to be made on an anaerobic plan, because naturally there is no air within the gall-bladder. It, furthermore, is possible that, with the ever-shifting fluid within the bladder itself, the bacteria may be found only here and there, and also may vary, and just that particular bit of bile that is used for making the culture may not contain any. In order to obtain more reliable results, we have therefore adopted the following plan:

We always have a competent assistant of the pathological laboratory at the operating table. Immediately after aspiration, he receives the syringe with the bile from the gall-bladder and spreads it on the agar plates. After the cholecystectomy, the viscus is bisected transversely; the cystic duct with the proximal half of the gall-bladder, is placed in a 5 per cent. formalin solution, so as to preserve the mucosa which otherwise for pathological examination rapidly undergoes autolysis, while the fundus with the distal half is used for tissue culture. Examination has repeatedly proved the presence of bacteria in the gall-bladder wall, though the turbid, greenish black bile from the bladder failed to show a growth. Proceeding systematically in this way, we hope gradually to gain data of value in post-operative laboratory examination—data which will allow of definite diagnostic conclusions on basis of pre-operative findings.

So far we have found that recurrent cholecystitis without stones is a much more frequent disease than is generally believed, that it seems to occur oftener in the male than in the female sex, that many patients with vague complaints of distress of the normal equilibrium in the upper abdomen, suspected to be referable to a gastric or duodenal ulcer, suffer from cholecystitis, often without stones.

Persistently recurring pains in the left side of the upper abdomen as well as in the region of the heart—so frequently diagnosed in the male as spurious angina—are also frequently due to a gall-bladder infection. I have seen a number of such instances. (Proceedings of N. Y. Surg. Soc., 1919, *Annals of Surgery.*) Complete gastric analysis and radiography of stomach and of the left kidney for stone had given a negative result in these cases, the urine, too, having been found normal. But examination of the duodenal contents gave the right clue, and was corroborated by operative findings. These pains on the left side of the abdomen are more often associated with cholecystitis and cholelithiasis than with cholecystitis without stones.

Of course, not too much must be demanded from this mode of examination. It cannot throw definite light on the question whether the turbid bile is discharged from the gall-bladder, or from the liver and bile ducts—at least not thus far.

Perhaps the further working out of the problem will permit us to do this also. I would but refer to Meltzer's proposition to instil a solution of magnesium sulphate into the duodenum in order to stimulate the discharge of bile.

However, the method will, I believe, be of importance in clearing up the cases of recurrent colicky pain after cholecystectomy. These pains do not necessarily always depend on the gradual descent of calculi, or of "liver gravel" found within the branches of the hepatic ducts or a spasm of the constrictor muscle at the papilla of Vater, but may depend on the presence of a persisting slight subacute or chronic cholangitis, a condition which the duodenal tube may greatly assist in proving.

With reference to the surgery of the gall-bladder, but a few words:

1. So far as indications point today, it appears to be better surgery to extirpate the infected gall-bladder without stones than to drain or leave it in place. Such a bladder, stitched to the abdominal wall, though at first drained for quite some time, may in the future come to represent an infected focus from which the system is intermittently flooded with toxins. If secondary cholecystectomy is refused by the patient, chronic pancreatitis may develop in addition to the chronic recurring cholecystitis, and invalidism may develop for the remainder of life.

It is also most likely that the chronic cholecystitis *without stones* which often shows bacteria suspended in its bile and habitating within its walls, is to be considered "the precursor" of chronic cholecystitis *with stones* in many instances.

2. I prefer operating from the fundus toward the cystic duct, in spite of the somewhat increased hemorrhage from the gall-bladder bed. In this way one will always be ready properly to meet any of the anomalies so often present near the junction of the cystic and the common ducts, so beautifully demonstrated by Eisendrath's specimens. One will also be able always to ligate the cystic duct very close to the common—an important point, inasmuch as by so doing nature's effort to distend the stump and form a miniature new gall-bladder with all the inherent faults of the original larger one will be forestalled.

3. The results of cholecystectomy are almost as good as those

of appendectomy. Of 100 patients operated on we have seen practically 100 get well. Something extraordinary, something unforeseen, beyond the control of the attending surgeon, has to happen to have the operative result disappoint expectations.

4. To close the abdomen completely, without drainage, as in appendectomy, seems inadvisable; a leak may occur at the tied stump of the cystic duct, permitting infected liver bile to pass into the peritoneal cavity, and thus cause death from septic peritonitis after an original straight recovery. Drainage is more cumbersome but meets all emergencies. The patient's safety is the first law.

5. The principal advantage accruing from the preliminary more refined diagnosis by means of the examination of the duodenal contents, for the patient as well as for the surgeon, is the indication derived from it with reference to the place of the incision. Every surgeon, I am sure, has often felt dissatisfaction on cutting down on the appendix and then introducing the hand into the abdomen and palpating the gall-bladder region. When using the intermuscular (McBurney's) incision, the hand cannot be introduced, and the para- or per-rectus cut must be made long enough to permit the hand to pass. Having the fingers in place, we can but determine the presence or absence of calculi in the gall-bladder; we cannot clearly feel pericystic adhesions, omental attachments, etc. When we know, however, before we proceed with the operation, that the bile is turbid, it becomes our duty to cut down on the gall-bladder and then pull up the cecum with the lower portion of the ileum into this wound, and remove the appendix. Then we are able to view the gall-bladder and its immediate surroundings; we can inspect stomach, duodenum and liver, can palpate the head of the pancreas, common and hepatic ducts—in other words, can make the usual survey of the upper abdomen, the same as we are wont to do in other gall-bladder operations. Finally we can aspirate the gall-bladder and find out the quality of its contents, comparing it with that of the bile examined before operation, and on the basis of this finding determine the necessary operative procedure.

It is interesting to note how the whole method of examination before the operative procedure, as described above, has changed the indication formerly derived from the results of inspecting and palpating the gall-bladder. Formerly a non-glistening, whitish and thickened bladder gave the signal for ectomy; a shining, bluish, soft gall-bladder was left in place. Today the color of the wall, whether bluish, pink or white, its reflex and softness are no longer determining factors for attack or non-interference. The *quality of the bile,* determined systematically with the help of the duodenal tube, *previous* to operation, and the condition of the bile aspirated from the gall-bladder *during* the operation, guide the surgeon in his decision of where to incise and what to excise.

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DISCUSSION.

(See page 67.)

THE NEED OF EARLY DIAGNOSIS AND TREATMENT OF CHOLECYSTITIS AND CHOLEDOCHITIS.

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PHILADELPHIA.

There are probably no six inches of the entire alimentary canal in which states of organic disease are so prone to develop as in the first and second portions of the duodenum; nor is there any zone into which the elements of differential diagnosis enter in a larger and, at times, more perplexing manner. This, the hot bed of digestion, has emptying into it the mixed or mixing secretions from the stomach, the liver, the gall-bladder, the pancreas and the secretion from the duodenal mucosa itself.

The physiology of the digestive secretions in normal people from these various sources has become better understood during recent years. The pathological physiology of states of disease in this zone has been the subject of much profitable investigation during a still more recent period. Much light has been thrown upon the subject by means of carefully conducted animal experimentation. The more widespread use of the duodenal tube in the hands of capable students of gastrointestinal disease is contributing greatly to our knowledge by clinical experimentation on human beings, both normal and those suffering from disease. We have learned how to interpret our findings in the duodenum much more clearly and accurately; we can quite easily determine states of duodenitis and can differentiate those that are catarrhal, those that are infected, and those which show unusual exfoliation of dead and dying epithelium; we can feel reasonably sure of separating our more superficial erosive states from those of true ulceration simply because we are gradually training ourselves to make better use of the materials recovered by means of the duodenal tube for more painstaking cytological, bacteriological and chemical studies.

Differential diagnosis has been gradually extended so that we are now fairly sure of the soundness of our investigations into pancreatic states of health or disease, although there remains a very great deal of work to be done in this field. We have made, too, considerable progress in our ability to diagnose accurately many of the states of disease of the biliary system. But, unfortunately, most of our fruitful efforts, as in the cancer problem, have resulted in the elaboration of various methods and various tests that concern themselves in the proving of disease already well established.

Furthermore, our methods of diagnosis have been more largely indirect than direct. We have learned the value of the carefully taken and searching inquiry into the presenting symptoms, we have learned to interpret more clearly the transition of the earlier symptoms into those that in themselves are almost diagnostic, we have extended the scope and the accuracy of our methods of physical examination, and our eyes and our fingers have gradually been trained to take cognizance of more minute abnormalities than would have been thought possible a generation ago. Much of this has come about through the pioneer efforts of the surgeons, who have taught us by object lessons in living pathological anatomy at the operating table, the correct interpretation of historical syndromes and of data gained by physical examinations.

We have made great progress, too, in the art of diagnosis of various biliary diseases, as we have caught the importance of focal infection and its march from primary to secondary fields of activity. By the more recently accepted methods of examination of blood chemistry we have learned the significance of an increased amount of cholesterol in the blood serum; we have connected some of the clinical links regarding the incidence of pregnancy, tight lacing and other conditions with gall-bladder disease, especially in relation to the formation of gall stones. As a more direct means of diagnosis we have turned to the roentgenologist for the important aid he can now furnish us with his positive and negative shadows of formed calculi or of increased connective tissue formation in the wall of the pathological gall-bladder. But direct as is the evidence given by the X-ray, it fails us, perhaps, in half of our cases, and even when supplied serves only to prove a pathological state already well established. In other words, the greater part of our diagnosis of gall-bladder problems, thus far made practical, supplies us with information pointing to disease so fully developed that we have been handicapped in applying methods of treatment which, to be successful in ultimate cure, have become more and more radical.

The field of treatment by almost common consent has fallen to the surgeon because our accepted methods of medical management have woefully failed to bring results other than palliative.

For a little over three years I have taken great interest in developing a more direct means of differential diagnosis of diseases of the biliary system which lends itself admirably not only to the direct detection of organic disease well established, but also gives promise of a better understanding of functional disorders of the liver and gall-bladder and the recognition of pathological physiology which may act as part of the precursory states in the development of the later full blown disease.

We have known for some time that it is possible to drain bile from the common duct and from the liver and collect it by means of the duodenal tube for examinations that have been directed largely to the estimation of pancreatic efficiency. (Einhorn, Gross, Crohn.) But a great step forward was made when Meltzer suggested to us a means of making the gall-bladder contract and discharge its contents. This has opened an entirely new field of clinical diagnosis and investigation and has widened the horizon of our vision for the recognition and correction of the early states of disease of the gall-bladder and ducts that may ultimately lead us to the goal of present-day medicine, namely, the prevention of another group of diseases which has claimed a heavy toll of suffering and death. I allude to gall stones and serious late states of infection of the gall-bladder, liver and its ducts.

Meltzer (1), in an excellent article giving his rational conception of the physiology of the filling and discharge of bile from the gall-bladder, as governed by his law of contrary innervation, appended a little footnote to the effect that he found that solutions of magnesium sulphate, when locally placed in the duodenum, without first passing over the gastric mucosa, would cause a relaxation of the tonus of the duodenal wall and would thereby relax Oddi's sphincter of the common duct and permit the discharge of bile into the duodenum.

Immediately after the publication of Meltzer's paper, in April, 1917, I was able to demonstrate that the use of magnesium sulphate, locally, in solutions of various strengths, in the duodenum of human beings would very promptly deliver bile through the duodenal tube in varying quantities and of varying quality. It would do this when the duodenum was previously bile free, indicating that the magnesium sulphate had relaxed the sphincter action of Oddi's muscle. Further than this it was noticeable that the character of the bile recovered by means of the duodenal tube underwent certain definite changes in color and viscosity, first a light lemon to golden yellow, then a deeper, richer, more syrupy golden yellow, finally changing to a very uniformly light lemon yellow, thinner and less syrupy than either of the first two; and that this sequence occurred in all normal cases.

It was not long, however, before I examined a patient suffering from symptoms strongly suggestive of biliary disease in whom the second sequence of delivery of the deeper golden yellow bile was replaced by the recovery of over five ounces of deep greenish black bile very viscid, almost tarry. What did this mean? Where was this bile coming from?

The natural inference was that it was coming from the gall-bladder. But could it be really possible to drain the gall-bladder by magnesium sulphate and the duodenal tube and get it out in a bottle? Yet the cytology of this bile microscopically revealed mucopurulent particles rich in pus cells, large masses of deeply bile stained columnar epithelium, inflammatory debris, masses of bile crystals, and was simply swarming with bacteria, chiefly cocci. Culturally the latter turned out to be Streptococcus viridans. The patient was operated on ten days later and the gall-bladder found to contain bile of the same black color and viscosity and Streptococcus viridans was isolated from the bile.

This case and several that had preceded it were the starting point in the use of a method which I first described in a paper (2) and published seven months ago after I had made more than a thousand observations of the practicability of a nonsurgical method of biliary drainage. With certain exceptions, to which I shall later call your attention, it is possible to drain the gall-bladder wholly or partially of its fluid contents; to drain the bile ducts and to obtain bile freshly secreted from the liver cells. Furthermore, it is possible to segregate these various biles from their numerous sources by collecting them in individual bottles for chemical, microscopical and bacteriological examinations that give us a direct method of differential diagnosis between various diseases of the biliary system.

In the direct evidence it furnishes us, it far surpasses any diagnostic method yet available, and materially assists our correct interpretation of the presenting history, the physical examination, and the information furnished by the Roentgen ray and by the laboratory examinations into the state of gastric chemistry and motility, and of the stools, urine and blood chemistry. But most important of all, it furnishes direct diagnostic evidence of the beginnings of biliary stasis, of masked focal infection that precede the more florid states of biliary disease and give rise later to the symptoms, the physical and laboratory findings that are usually so clear cut as to make a tentative diagnosis of gall-bladder disease quite tenable and to warrant the dictum, "We will do an exploratory operation and find out what the trouble really is." This is all very well for the doctor, but a little rough on the patient if there is another reliable and direct alternative method available. In other words, we must learn how to find the direct evidence in the early cases exhibiting the chronic but vague dyspeptic symptoms and not leave it to the exploratory operation to decide whether the trouble lies in the upper right or the lower right abdominal quadrant. Even with the stomach, duodenum and gall-bladder well exposed the surgical eye and finger often fail to detect the presence of an early cholecystitis, choledochitis or duodenitis (usually the forerunner of ulcer), because there is no recognizable gross pathological change (quite ignoring the pathological physiology that precedes gross pathology), and the appendix is then removed usually because it presents a sufficiently pathological condition to warrant it, but not infrequently it is quite innocent and is removed simply because the abdomen is open and it doesn't increase the risk of the operation.

What is the result of this? If there is present concomitant disease of both appendix and gall-bladder, as Rosenow's work on streptococci leads many to suspect, and if the gall-bladder is harboring streptococci, but in a state of masked focal infection, not severe enough to cause diagnostic symptoms with a parallel gross pathological condition, but nevertheless sufficient to produce a pathological biliary physiology and a positive bacteriology to be found by him who looks, the result is this. The surgeon explores, and finds no upper abdominal pathological condition, no enlarged glands, no stones, no adhesions and the gall-bladder expels its contents under forcible digital pressure (but can it do so under its own muscle power?), and because there is no gross pathological condition the surgeon says everything is normal here, leaves a gall-bladder harboring streptococci, and proceeds to account for the symptoms by removal of the appendix. The patient gets well—that is to say, he recovers from the operation—his symptoms improve temporarily, aided by his hospital rest and the removal of his appendix, provided it was in a truly pathological condition; but usually between 6 and 24 months later his symptoms recur, progress in frequency and severity, and change in character until finally the clinical picture of full blown gall-bladder or duct disease presents itself and, in the judgment of most doctors, operative interference again becomes imperative.

This is not to be wondered at, for it doubtless is true that operative interference is the best procedure at the present time in the properly skillful hands. The surgeons have been successful pioneers in the field of gall-bladder therapy because the indirect efforts of the internist with his cholagogues and bile disinfectants, his medicated waters, his diets and his prescription to attend expensively famous foreign spas have been inadequate and uncertain, whereas the direct attack by the aseptic scalpel is productive of prompter results whether good, bad, or indifferent. As Dr. John B. Deaver (3), so apt always in his quotations and epigrams, says in a recent paper, "If thy right hand offend, cut it off." But let us pause a moment and consider. Of course, it is easy for the skillful surgeon to cut it off, but it is quite another matter to put it on again if the first experiment doesn't work. It is one thing to remove with impunity the appendix which possesses no (or an unknown) function (although many an innocent one has been removed in the past, as have healthy tonsils and teeth during their respective crazes), and quite another thing to remove ruthlessly and routinely every gall-bladder because some harbor streptococci in their lymphatic tissue and in their walls. As I have said, it is all very well with the patient if it works. But suppose—and we know that this often happens, for the surgeons tell us—suppose the common duct remains infected after surgical drainage is completed and later becomes obstructed, what happens then when the distensible reservoir for liver bile has been removed? The safety valve has blown off. The common duct dilates and vicariously tries to assume the duties of the gall-bladder; diverticuli may appear, duct bile become static, new concretions form, and sooner or later the secreted bile dams back into the liver and biliary cirrhosis has begun. Deaver's biblical quotation is apt, but the title to his paper, "Operation and Reoperation for Gall-Stone Disease," is still more apt; besides, the mortality table is not published.

Perhaps if the careful student of internal medicine adopts the motto, "Search and ye shall find," it may eventually be better for the patient, although the work may be slow and laborious and lacking in spectacular brilliance. One has only to peruse some of the better recent papers on gall-bladder surgery to realize that operation means facing undeniable risks. Although the mortality has been steadily reduced, it was nearly 6 per cent. in the thousand cases recently analyzed by Smithies (4), with 35 per cent. of associated pathological lesions of the upper abdomen found at operation (enlarged lymphatic glands, acute and chronic pancreatitis, enlarged liver and peptic ulcer), indicating late diagnosis with well established pathological conditions. Added to this are the complications pictured by the surgeon, the skilled full-time operator and not the occasional surgeon, of damage to the hepatic and common ducts, the recurring adhesions, the persistent fistulas, the occasional fatal bleeding from the liver or from an accidentally torn blood vessel, the occasional traumatic puncture of the gut or the spilling of infective streptococci bile with resultant peritonitis, to say nothing of Nature's recurrent complications of new stone formation in dilated common ducts again obstructed, necessitating recurrent operations, and we have a true picture of the gall-bladder problem as it stands in the light of our present methods of diagnosis and treatment. Certainly it is far better than it used to be, but is it as good as we can make it?

Description of the Method.

In order to present the method which I hope can be proved in other hands to possess the merits of early or late, direct diagnosis in gall-bladder and duct disease, and of potential merit in the treatment of selected patients suffering from these diseases, I must go briefly into the fundamental principles which underlie the method. Much of this has already been presented in four previous papers on the subject (5, 6, 7, 8).

The biliary system consists of a constantly secreting organ, the liver, passing its secretion and excretion, the bile, down a series of tubes guarded at their terminal outlet by a muscle possessing a sphincter action. Placed between the liver and Oddi's muscle sphincter is the gall-bladder with elastic walls permitting varying degrees of physiological distensibility, to act as a reservoir for excess bile secreted during the periods when the duct sphincter remains closed. Thus we have a mechanism that physiologically consists of the elaboration of a constantly secreted fluid, which, however, is discharged intermittently.

Upon what does the mechanism of partially or wholly emptying this biliary system depend?

Meltzer's law of contrary innervation (9) as he applied it to the filling and discharge of the gall-bladder was briefly to the effect that the sphincter of the common bile duct and the muscles of the gall-bladder were supplied with inhibitory and motor nerve fibers from the splanchnic and vagus nerves which acted antagonistically to one another. That when the inhibitory fibres relaxed the tone of Oddi's muscle at the sphincter of the common duct, the motor fibres to the gall-bladder caused its muscle to contract and therefore discharge its stored up bile into the duodenum until such time as the sphincter would contract again, when, automatically, the inhibitory fibres to the gall-bladder would cause a relaxation in the gall-bladder wall, thus preventing a further expulsion of its bile, and it would then resume its passive rôle of acting as a reservoir for the bile freshly secreted from the liver. Meltzer pointed out that the normal physiological stimulus to produce biliary discharge lay in the character of the food chemistry which passes through the duodenum. To establish this he quoted the experimental work of Bruns, which showed that normally no bile appeared in the duodenum as long as the stomach was empty, but that the entrance of a food chyme into the duodenum was the signal for the ejection of bile from the common duct. He further quoted the experiments of Rost, who proved that injection of peptone or albumosis through a duodenal fistula in a normal dog immediately caused a discharge of bile from the common duct and proved that this took place by a reflex act which caused a contraction of the gall-bladder and simultaneously a relaxation of sphincter of the common duct. Furthermore, Rost had previously established the fact that after animal cholecystotomy the escape of bile through the papilla of Vater became continuous, whereas in normal animals it was discharged intermittently. This argued strongly in favor of Meltzer's law of contrary innervation in the fact that simple cutting into the wall of the gall-bladder would destroy the antagonistic action of the nerve supply to the gall-bladder and common duct sphincter. This mechanical breaking of the nerve circuit by operation can be easily demonstrated in postoperations in which the gall-bladder has been either opened or removed, namely that bile is being discharged continuously into the duodenum so long as the common duct remains unobstructed. Furthermore, I believe this break in nerve conduction is mimicked in disease involving the wall of the gall-bladder or in the wall of the duodenum adjacent to Oddi's muscle (duodenal ulcer, duodenitis, duodenal adhesions), because in this type of disease I frequently find continuous discharge of bile into the duodenum with a reflux of grossly recognizable bile in the fasting stomach in early as well as late pathological states of the duodenum and of the gall-bladder. This observation of what is certainly pathological physiology appears to me to be a very important diagnostic factor in itself and useful because it may be indicative of early changes. The significance of fasting and digesting biliary regurgitation will be the subject of a future communication.

In regard to magnesium sulphate: Although Meltzer did not specifically state in his footnote that it would cause expulsion of gall-bladder bile, but only that it would relax the duct sphincter, the inference was plain that if his law of contrary innervation was sound, anything which would cause inhibition of tonus of Oddi's muscle must, *ipso facto*, cause contraction of the gall-bladder musculature. This is not so. Yet it is fortunate for the progress of this work that Meltzer was experimenting with magnesium sulphate, for it will call into action this antagonistic or reciprocal action of duct sphincter and gall-bladder.

But there are other substances (benzyl benzoate, belladonna, potassium permanganate) that will relax the duct sphincter and yet will not produce expulsion of gall-bladder bile. Similarly there appears to be a selective gastric food chemistry that will electively cause expulsion of gall-bladder bile in large quantities on the one hand and discharge of pancreatic secretion on the other. For instance, as Rost has already experimentally shown, peptones and albumoses (end products of acid gastric digestion) will call forth a richer and larger quantity of bile in the duodenum. This is seen in the proteid and fat test meals. Whereas, a carbohydrate meal, although bathed in the same acid gastric juice, will call forth more pancreatic juice and little, if any, gall-bladder bile; although naturally the bile in the common duct and that secreted by the liver is being discharged during the time that pancreatic secretion is being poured out. This appears to support the accepted theory of the physiology of automatic (or reflex) discharge of digestive secretions or enzymes according to the chemistry of the food stuffs to be digested. What the exact character of this mechanism may be, whether nerve reflex or blood reflex, or true harmonic action, or a mixture of them, will require further investigation both on animals and on human beings. But when we remember that the pancreatic duct and the bile ducts in 90 per cent. of anatomical subjects discharge their contents through a common ampulla governed, apparently, by the same sphincter, in each case the sphincter itself must relax to permit such discharge, yet the gall-bladder may not necessarily contract each time, certainly not with the same degree of vigor. So it appears that while Meltzer's theory of the physiology of filling and discharge of the gall-bladder bile is thoroughly worked out, and while his law of contrary innervation is substantially sound as regards magnesium sulphate (perhaps in this direct duodenal action a true hormone for gall-bladder contraction), nevertheless there are certain substances, while they relax the common duct sphincter, have an elective action on the gall-bladder or on the pancreas individually, and no doubt certain substances may have a dual action.

A great deal of this problem of physiology remains to be worked out before we can get away from a certain empiricism in the use of various diets and various drugs. The method of direct clinical investigation in health and disease by the duodenal tube, using various chemicals and food chymes, opens up a most attractive and profitable field of work.

To return to the subject of this paper. The method that I have suggested permits of making direct observations on the bile obtained from the several sources in the biliary tract.

To make possible accurate diagnosis of the duodenal biliary zone it is necessary that we adopt means to prevent cytological and bacterial contaminations from the mouth, teeth, tonsils, respiratory tract and stomach from confusing us in our interpretation of duodenal and biliary materials. To avoid this as far as possible I have adopted the following routine method in diagnosis: The use of proper apparatus will aid in the performance of good work. The patient presents himself with a 12-hour fasting stomach. He then brushes his teeth carefully, rinses and gargles his mouth and throat thoroughly, first with a strong solution of potassium permanganate (one grain to the ounce), then with a mildly astringent solution of zinc chloride. The duodenal tube which has stood over night in a 2 per cent. solution of lysol is freshly sterilized by boiling and is passed to the stomach. The fasting residue is aspirated and set aside for chemical cytological and bacteriological examination for comparison with the findings later recovered from the duodenum. The stomach is then rinsed to sparkling clearness, using gravity douching from 250 c.c. irrigating tanks or syringe douching, and recovering the wash water in 250 c.c. conical graduates in which can be noted how clean the stomach is, mucus, shreds, mucopurulent plugs, and other material which microscopically yields much valuable information. After the wash return is sparkling clear the stomach is made astringent with a zinc chloride solution (layoris), and then rewashed thoroughly. It is surprising to observe how often a stomach apparently washed clean, after being made astringent, will press out from the mucosal tubules mucopurulent masses which plug the ducts and which, microscopically, show, in true gastritis cases, masses of gastric epithelial cells infiltrated with small round cells and polynuclear leucocytes and often swarming with bacteria. It is to be noted that none of this epithelium is ever bile stained. After the use of the astringent and washing clean again, the stomach is then disinfected with 250 c.c. of potassium permanganate, one to ten thousand, which is immediately recovered and the stomach again washed clean to crystal clearness. This requires about 20 minutes to accomplish. This, so far as it is possible, prevents contaminated material from the upper zones confusing our interpretations of material later obtained from the duodenal biliary zone. After diagnosis has been completed and local treatment has been instituted it is not so necessary that preparation of the mouth and stomach should be so carefully carried out except when indicated in patients with dirty mouths and dirty stomachs. A little water is then left in the stomach to encourage peristalsis, the patient lies down and turns on his right side and very slowly swallows an additional 20 cm. of tubing to a total distance of 75 or 80 cm. from the teeth, according to the length of the thorax. I insist that they take 20 minutes to swallow the 20 cm.; slow swallowing at this point is often the secret of rapidly entering the duodenum. The duodenal tube is then connected to the first sterile aspirating bottle and the duodenal secretion is aspirated to note whether the common duct sphincter is normally closed. The duodenum is then douched with about 75 c.c. of a 33 per cent. solution of magnesium sulphate. This I believe to be the optimum strength for good sphincter relaxation and gall-bladder contraction. Where the gall-bladder is found atonic it is sometimes necessary to restimulate its contraction discharge by douching again with half the amount of magnesium sulphate solution.

Before the magnesium sulphate has entirely run in, the tubing is connected to the bottle and gentle aspiration started and the magnesium sulphate returns at first uncolored, but within one to six minutes, normally, the sphincter is relaxed and the magnesium sulphate becomes tinged with bile, which becomes steadily deeper until pure bile alone is being recovered. Another bottle is then attached and observations are continued through the glass cannula window inserted in the tubing about eight meters from its proximal end. When the first bile, which I call A bile and believe to be that contained in the common duct plus a few drops from the cystic duct and a few mils, perhaps, of freshly secreted liver bile passing down the hepatic ducts, deepens to a distinctly deeper golden yellow or becomes in any way off color and more syrupy and of heavier viscosity, this bottle is detached, another quickly attached and drainage of this darker bile allowed to continue until the third transition to a very much lighter yellow and thinner bile appears, when a final bottle is attached to continue the bile collection to the end of the drainage period.

The darker bile appearing in the second transition I call B bile and believe it to be derived almost entirely from the gall-bladder, mixed, of course, with a few drops or mils of liver bile. My reasons for believing this bile to be gall-bladder bile I have given at some length in a previous paper (8). The third type of very light yellow limpid bile which appears in normal cases in the third transition I call C bile and believe it to be bile recently elaborated by the liver cells and freshly secreted. It has invariably appeared at the termination of each drainage in nearly 2,000 observations which I have made up to this time. Of course, one cannot hope to segregate these several biles absolutely unmixed with the other, but if one is careful in segregating them it is surprising how accurately they can be separated with a little practice, and I feel safe in saying that, if carefully done, the majority of A bile is common duct bile, that by far the majority of B bile is derived from the gall-bladder, and, if the latter has emptied completely, that practically all of C bile is freshly secreted liver bile.

DIAGNOSIS.

Diagnosis is then developed around the direct study of the bile and the manner of its discharge—the promptness with which A and B biles appear, the amount of B bile and the steadiness or the intermittency of its discharge, suggesting normal tonus, subtonus or hypertonus of gall-bladder musculature and giving inferences as to its capacity; on the gross appearance of the several biles, color, consistency, viscosity, transparency, turbidity, flocculations, mucus, etc., and especially the careful examination into the cytology (epithelium, whether bile stained, its source, pus, leucocytes, crystals, concretions, red blood corpuscles, inflammatory debris, mucus, bacteria); into the chemistry (lecithin, cholesterin, calcium, pigments, effervescence on acidification); and into the bacteriology by culturation of each of the segregated samples of bile.

The bacteriological examination must be carefully conducted and promptly done to prevent streptococci and other less hardy organisms becoming overgrown with the more rapidly growing colon groups, Bacillus pyocyaneus and Bacillus subtilis. Cultures should be made at the time of the withdrawal of bile in the office, clinic or hospital, and planted in glucose broth flasks, blood agar tubes, and a third sample put in a sterile test tube, labeled and promptly sent to the pathologist or bacteriologist unless you are qualified to do the work yourself.

I have learned that I get more reliable cultures from planting the mucopurulent flakes, especially when heavily bile stained, which sink down to the bottom of the bottles, particularly those of B bile. These mucopurulent flakes, lifted out by a sterile pipette, are representative of material from the floor or walls of the gall-bladder, ducts, or duodenum. Microscopically they show by far the most interesting and conclusive cytological condition. If more cultures were taken at operation from the gall-bladder, from the mucopus from the floor of the gall-bladder, and not simply from the supernatant bile, I believe the average of positive cultures would be much higher, whether the gall-bladder showed gross pathological changes or not. Withdrawing bile by a sterile hypodermic needle and syringe often gets the supernatant bile only. I wish to emphasize the need for careful cultural technic and prompt examination. Much important differential diagnosis hinges on this.

Again I would like to point out that it may be possible to decide where the source of the maximum infection may be, even though A and B, or A and B and C biles all deliver, say, streptococcus and colon bacillus, by taking advantage of colony counts. For instance, if you plant loopfuls of A and B biles and sow them through blood-agar petri plates and find that A bile grows 7 colonies and B bile 94, it is reasonable to conclude that the major source of the infection is the gall-bladder and not the duct. Similarly, if the colony counts from C bile are far larger than A or B, the liver is to be suspected of being infected.

This plan is working out well and checking up well in differential diagnosis. I do not see now, however, how we can ascertain definitely whether or not the wall of the gall-bladder or of the ducts, or the duodenal mucosa, is definitely infected beyond the possibility of recovery by free drainage and topical treatment and sensitized vaccines. These direct diagnostic findings can, therefore, be used to amplify or to interpret the information secured from the history, the physical examination and from special examinations, such as the X-ray, stool, stomach and blood.

We can thus hope progressively to modify in the future the one time true statement of Stockton (10): "It is difficult to reach a clinical knowledge as to the amount of bile that is being passed and as to the various constituents of the bile," and the statement of Smithies (11): "The average textbook considers cholecystitis in a vague, uncertain way, as though it were not an ailment second in frequency to all intraabdominal diseases only to lesions of the appendix. Commonly cholelithiasis meets recognition as an acute dramatic, abdominal crisis, in which the chief rôles are played by colic, chills, fever, sweats, and jaundice."

The differential diagnosis between choledochitis and cholecystitis depends to a large extent on the bacteriology and cytology plus the gross normality or abnormality of the bile. This will be referred to in greater detail when the diagnosis of atony of the gall-bladder is discussed. Of course, if the gall-bladder has previously been removed, the problem is easier. Empyema of the gall-bladder is easiest to diagnose directly, provided the gall-bladder is mechanically able to discharge a specimen of its contents. Dr. Brown, of Montana, recently told me that he had examined some 70 or more suspected gall-bladder cases by this method. Among them were four cases of empyema successfully and directly diagnosed and two of the four he had successfully drained and tided over acute complications that did not warrant the risk of surgery at the time.

CHOLELITHIASIS.

Regarding the diagnosis of cholelithiasis, some helpful points can now be suggested. Of course, the recovery of gall stones themselves is the *sine qua non* of this diagnosis. I have recovered small concretions through the duodenal tube in one instance, and on several other occasions have made stones pass either out of the gall-bladder or out of the duct, stones too large to be recovered by tube but found on sieving the stools. In none of these cases, however, do I feel that this would have happened at the time of diagnostic drainage if magnesium sulphate introduced locally did not possess the power to relax the sphincter and to contract the gall-bladder wall. Why it loses much of its power to do so if first passed across the gastric mucosa, as Meltzer first noted and which I have confirmed, I cannot explain, but such, apparently, is the case.

Next in importance to direct recovery of definite gall stones, gall sand and the sense of grittiness to the finger suggest the calculus forming possibility. So does the microscopic finding of large agminated masses of precipitated crystals of bile salts or pigments, since it suggests that the liver cell has lost the power to hold these substances in solution, as occurs in the formation of liver or hepatic duct stones, or that the bile in the gall-bladder has become so static that excessive concentration and crystallization has taken place. I have previously shown that the sudden dense turbidity that one sees taking place in an otherwise perfectly transparent bile during a drainage is due to a sudden spurt of acid gastric juice entering the duodenum and mixing with the bile. This was confusing at first and is still annoying. Dr. Bartle, working with me, found that this turbidity could be artificially produced in the case of every clear bile by artificially adding dilute hydrochloric acid. The turbidity varies according to the strength of the acid and the chemical constituents of the bile. Later certain clear biles were encountered in which an effervescence as well as the turbidity was produced on adding hydrochloric acid, similar to the reaction of acetic acid and calcium carbonates in the urine, and the question has been suggested as to whether this might mean the possibility of potential or formed calcium carbonate stones in the gall-bladder. More work must be done on this point.

Atony of the Gall-Bladder.

Relative atony of the gall-bladder is something I believe we can diagnose and which I consider to be of extreme importance because I believe it to be one of the earlier phases of gall-bladder disease and the forerunner of gall stones and of gall-bladder infections. This diagnosis is suggested in three ways:

1. The recovery of static or off color bile, ranging from the deeper shades of golden yellow, into the green yellows, green blacks, and blacks, and possessing an increasing viscosity from that of a thick syrup to that of tar. Where the viscosity is heavy and the cytology shows much mucus and desquamating masses of bile stained, high columnar epithelium, and quantities of precipitated crystals, I consider this an atonic catarrhal cholecystitis and a potential forerunner of calculi. I have seen this type alone as well as the type of infected cholecystitis with a swarming bacterial flora and pus, blood and inflammatory debris. This is the outspoken type giving rise to well marked clinical symptoms. But I have also frequently seen the masked infective cholecystitis with swarming bacteria and static bile, but no cytological inflammatory reaction or marked cellular destruction. These are the cases that are early, do not show interpretable clinical symptoms,

but give rise to the vague atypical dyspepsias, and these, too, are the cases which operatively are passed over as grossly normal and in which the appendix is removed and the masked focus left to breed pathological conditions.

2. In the amount of static bile recovered. If a gall-bladder's normal capacity may be considered two and a half ounces, and if four ounces of this type of bile can be recovered in bottles, it seems reasonable to assume that the gall-bladder in question must be functionally atonic and unable to move its contents promptly or the cystic or common ducts must be partially obstructed. If 6 to 12 or more ounces of this static bile is recoverable (as in my series of cases), it must appear that the normal distensible sac has been overdistended, has become dilated and has perhaps ruptured some of its muscle fibres and may be progressing to an absolute atony. The functional type of relative atony seems to fit in well with many of the cases presenting symptoms of so-called biliousness and of cyclic migraine attacks. These also are groups that may be the forerunners of gall stones and pathological gall-bladder conditions.

3. In normal cases when B bile is recovered it comes continuously until replaced by the appearance of C bile and averages from one to three ounces, and further stimulation with magnesium sulphate fails to recover any more. Whereas, where atony is suspected, B bile appears, the bile is static to varying degrees, but gall-bladder discharge may be intermittent; that is, 2 or 3 ounces of B bile and then 10 to 30 c.c. of C bile and again 2, 3 or 4 more ounces of static B bile. Furthermore, it is possible to deliver more of this type of bile on restimulating with magnesium sulphate. It is reasonable to suppose that such gall-bladder musculatures are deficient in tone and incapable of emptying completely, as in atony of the urinary bladder, with its residual urine. Of course, there are limits to the amounts of magnesium sulphate that should be used. I think a safe limit might be placed at 90 c.c. of 33 per cent. representing 30 c.c. of the saturated solution. My custom is to start with 75 c.c. and note how much I recover in the first bottle unmixed with bile. If I recover, say, 40 c.c. I can then restimulate to the amount of 55 c.c. additional and still keep with the limit of 90 c.c.

It may be as well to mention here the fact that in many of these cases we are draining highly infected material from the biliary passages and that some of this fails to be aspirated into the bottles and passes down the intestines, possibly to infect susceptible zones lower down. There are two logical ways to overcome this. First, by douching the duodenum with various disinfecting solutions, potassium permanganate, silver nitrate or possibly chloramine-T, and get back what one can. I personally do this, but do not advocate it for any one beginning duodenal work of this kind, for it has an element of risk, because it is by no means certain that you can get out again what you put in. Secondly, to hurry along the infective material as rapidly as possible through the intestines. To do this I always follow each biliary drainage, whether for diagnosis or treatment, with a duodenal enema. I prefer Ringer's solution, for its healing qualities, reinforced by a five-tenths per cent. or twenty-five hundredths per cent. sodium sulphate, depending upon how much magnesium sulphate solution has failed to be recovered. The total amount of the duodenal enema I keep at 250 c.c., introduced at 105° and require at least 20 minutes for its introduction. This is usually effective in producing a large fluid or semifluid bowel movement in from 15 to 90 minutes. Furthermore, no patient leaves my office without being given a cup of bouillon and some crackers. This tides them over the faintness of hunger and free intestinal evacuation.

I wish to refer now to the diagnostic inferences that might be possible in the failure to obtain B or gall-bladder bile. They are so obvious that they merely need tabulation.

It might indicate any of these possibilities: 1. Obstruction of cystic duct, by, *a,* stone or stones; *b,* adhesions or angulations or stricture; *c,* pressure from without, tumors, lymphatic glands, or, *d,* inspissated mucus, hydrops. 2. Gall-bladder contents, may be entirely calculi and no, or relatively little, bile. 3. Weakness of gall-bladder musculature atony, dilatation, too weak to move its fluid contents. 4. Tarry bile, ultrastatic, too thick to flow. 5. Fibrosis of the gall-bladder.

TREATMENT.

This method during the past three years has been successfully used in the treatment of all of the states of biliary diseases mentioned in the following paragraphs. Three years is too short a time to predicate an opinion as to the ultimate possibilities this method of nonsurgical biliary drainage may possess. Its principles are soundly established and are logical. Furthermore, one is able to gauge progress made by the improvement in direct objective findings in addition to the usual method of estimating clinical and symptomatic improvement. The ultimate criterion of a cure in the real sense is more nearly within our grasp.

We are mechanically applying the surgical principles of free drainage for infected sacs, tubes and tissues, of free drainage for catarrhal states of inflammation of various grades but without infection, of free drainage for gall-bladders that are atonic and contain static bile in which sooner or later there develop stones or a more serious pathological condition, and while applying surgical principles we are doing it nonsurgically and avoiding certain surgical risks. Besides this, and even more important, we are preserving tissue which may possess a power of recovery of function beyond our present conception. Patients suitable for this method of treatment should be selected. Its real sphere of usefulness lies in giving a direct method of treatment in early stages of disease, diagnosed early, before gross pathological changes have taken place. Removal of pathological tissue, of gall stones, etc., must be left to the surgeon. Our aim should be to learn better to diagnose the beginnings of these diseases and to institute promptly direct, rational and safe measures of treatment. We may legitimately hope that this method, if intelligently applied, may decrease the number of cases requiring serious and dangerous surgery.

TECHNIC.

The technic of treatment is not difficult and can be carried out by hospital internes and even by nurses after a little practice. It does not require the expert supervision of the highly trained specialist, although it is naturally better if such service can be secured. Here it differs at once from the necessity of procuring the most skillful surgeon for surgery of the upper right quadrant of the abdomen. This is not the field for the occasional operator. It is time that we recast some of our accepted views. To operate and have the patient live and to operate and make the patient well are two very different things. It is one thing to cut out pathological tissue and quite another to restore pathological physiology already existent, or that created or increased by the operative procedure.

The technic of this treatment is easy, but it is the skill in the general diagnosis and the technic of handling the minutiæ of special diagnosis that require the highly trained specialist, and the better his training in pathology and physiology, and the keener his enthusiasm for the use of the microscope, the test and culture tube, the more valuable will be his opinion.

Simple catarrhal jaundice may be treated very satisfactorily by this method. The duration of jaundice in the ordinary case may be cut in half and potential drainage to the ducts, gall-bladder and liver may be prevented. Recent papers (12) have shown what can be done in the treatment of this condition by this method.

Choledochitis and cholangeitis may be successfully treated in favorable cases, especially where there has not been any surgical interference. That means the relatively early cases. Even in late cases where there has been well established pathology and several preceding operations this method may give an unexpected brilliant result. This was evidenced in one young girl whose case I have reported at length (8). She had three major and one minor gall-bladder and duct operations performed in three years, with two further years of constant suffering and remittent exacerbations of choledochitis, finally culminating in a very severe and acute attack, with complete duct obstruction, chills, fever, sweats, high leucocytosis and toxemia, yet her condition, with its very serious aspects, responded splendidly to this method of attack, and today the girl is well and has remained free from any further exacerbation for nearly three years.

I have within the week seen a young woman of 26 who had her gall-bladder and stones removed 23 months ago; she had two weeks of surgical tube drainage and nearly four months of dressing drainage. She remained free from symptoms for just two months, when exacerbations recurred, and during the past 18 months she had had an equal number of attacks of severe colicky pain which two weeks ago culminated in chills, fever, and acute obstructive jaundice. Unless her common duct can be speedily unplugged dilatation of the ducts and biliary cirrhosis may develop, because her safety valve, the gall-bladder, has been removed. It is more difficult to unplug such a duct when the gall-bladder has been removed, because the contraction of the bladder supplies a good part of the *vis á tergo*. It is remarkable to what extent the gall-bladder can distend, as witness the case of the little girl at Johns Hopkins Hospital, recently reported upon, whose very distended gall-bladder contained nearly a litre of bile.

What I have said of choledochitis applies to cholecystitis, perhaps if anything more favorably. Especially so in the early cases, such as those complicating typhoid fever, and masked focal infections of the gall-bladder. This is the time to diagnose and to drain, nonsurgically, in such cases and not wait for the development of a full blown pathological condition.

Empyema of the gall-bladder has been successfully treated during its acute phases in patients who presented severe cardio-renal contraindications for surgery. That is to say, their gall-bladders have been drained successfully, the maximum source of their toxemia has been temporarily removed, and they have been tided over to a point where corrective surgery for the removal of the pathological condition could be more safely practiced. There is nothing to prevent the success of nonsurgical drainage in empyema, provided the cystic duct is patulous. This is by no means recommended as the method of choice, but as a possible alternative measure worthy of trial in selected cases presenting grave surgical contraindications.

Cholelithiasis remains entirely beyond the scope of this method, although stones have been made to pass through the common duct. Our efforts should be directed to the detection and treatment of the early states of pathological physiology and to the prevention of calculi. The method has, however, distinct merit as a postoperative follow up treatment to prevent the reformation of stones and to continue to drain, *ex corpore,* still infected bile beyond the limits afforded by surgical methods of drainage. This has been proved a successful measure.

The one field where this method can be strongly recommended is in the treatment of biliary stasis or faulty retention of gall-bladder bile. If more cases of biliousness were investigated by this method it would surprise many of you to find the gall-bladder atonic to varying degrees and unable to discharge its static bile. These are the patients in whom, if they are left to themselves and their cholagogues, a quarry of stones will develop. These patients do extremely well and it is remarkable to see their improvement in color, digestion and bowel function. They lose their lethargy and recover their sense of wellbeing. Many of these atonic gall-bladders are harboring pathogenic microorganisms, but still preserve sufficient mucosal resistance to prevent infection of their walls. This is the time to treat them energetically by frequent drainage. Bacterial identification should be carefully made and autogenous vaccines have an important place of usefulness. It is very important to search back for primary foci of matched bacteriology in the teeth, tonsils, sinuses, bronchial tract, stomach or duodenum and remove them. Many of these cases of biliary stasis are associated with various forms of migraine. Some of them respond almost miraculously to biliary drainage; others are very resistant, suggesting a different causative factor.

SUMMARY.

To sum up in a few words, this method has already achieved a position of importance in the diagnosis of biliary diseases. In the field of treatment it is certainly the method of choice for biliary stasis, gall-bladder atony, and in the early states of catarrh and infection. It may be found to decrease the incidence of stone formation and thus of cancer of the gall-bladder. It will decrease the tendency to damage the pancreas and liver. It may decrease the frequency of acute and chronic pancreatitis, of biliary cirrhosis and possibly diabetes. It may have a place as an alternative method of treatment for some of the surgical groups presenting operative contraindication. It certainly is useful as a postsurgical follow-up plan of treatment in many cases.

More time must elapse to prove its final evaluation. Quite true, but one must start somewhere. It has had a good beginning. It may go further. It is within my province to call your attention to it, and within yours to prove that it has the merit which I believe it to possess.

As to future possibilities, it offers an attractive opportunity for further direct clinical investigation into and the interpretation of: 1. What are the cholagogues? How do they act? *a.* By increasing liver secretion of bile or the velocity of its discharge? *b.* Do they empty the gall-bladder? 2. Precursory states and phases of gall stones and infections, *i. e.,* biliary stasis and atony. 3. Parallel studies on pancreatic secretion, velocity of elaboration of ferments and their discharge. What are the elective pancreatic secretogogues? Have they a place in the prevention and treatment of diabetes? 4. Extending the scope of chemical investigations into the composition and physical properties of bile.

Here are many usefully important problems awaiting solution.

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DISCUSSION OF PAPERS OF DRS. EINHORN AND MEYER AND LYON.

DR. H. W. BETTMANN, Cincinnati: Both papers mark an advance towards the accurate diagnosis of gall-bladder disease. Any new procedure, like that of Dr. Lyon, is to be welcomed warmly as opening up new possibilities of direct attack. It will require several years of study to determine accurately the absolute value of his method.

Our discussion will gain in clearness if we divide all our gall-bladder cases into three groups.

1. The cases of frank colic.
2. Cases of recurrent cholecystitis.
3. The dyspeptic group.

There is little trouble ordinarily in making a correct diagnosis in the cases of colic. Direct observation of the attack and the exclusion of the causes usually suffice to insure the right interpretation.

The group of recurrent cholecystitis is a type which should be (but often is not) recognized by the attending physician and is often overlooked by the specialist. The patients come to the specialist in the healthy interval, but they are observed by the regular physician during the acute attack.

Even a mild attack of catarrhal cholecystitis is recognized by physical signs, and a bedside clinician of experience can usually recognize it by the localized swelling and tenderness in the gall-bladder region, and often by actual palpation of the gall-bladder. When the patient comes to the specialist in the interval, these symptoms are often lacking. Another point is the mild leukocytosis, which also escapes the specialist. The relation of leukocytosis to gall-bladder infection has never been sufficiently studied. Pus may be present in the gall-bladder without producing leukocytosis because of the poor development of the lymphatic system of the gall-bladder. On the other hand, a mild acute catarrhal attack of cholecystitis may often have a white count of 10,000-12,000, which disappears in a day or two. It is the same with fever. Acute attacks often have a mild fever of 99° to 100° F., which should help the clinician to recognize an existing inflammation.

The third or dyspeptic group forms the widest field for the application of the new methods. We should not forget, however, that the flow of bile is subject to the widest variations due to physiological and nervous influences. One examination by the new methods may lead us astray. Nervous conditions may accelerate or retard the flow of bile. A railroad journey will often induce constipation and the stools are gray and poor in bile. Seasickness can almost entirely stop the flow of bile for hours. Certain neurotic states act in the same fashion. Dr. Neusser of Vienna laid special stress on the relation of gall-stone colic to certain nervous states.

Dr. Lyon appears to accept the conclusion that infection of the bile ducts and gall-bladder occurs by extension upward from the duodenum. I believe that the testimony against this view is rather conclusive. Catarrhal jaundice is very rarely followed by disease of the higher bile passages. Regarding Dr. Lyon's specific method, only a varied experimentation will show if it is the best one. In health, the entrance of food into the duodenum is the physiological stimulus to relaxation of the papilla and contraction of the gall-bladder.

Whether magnesium sulphate in 25 to 30 per cent. solution will prove to be the optimum reagent, the future must determine. Dr. Meltzer, in reading his paper, spoke of peptones and albumoses as being the real stimulants to relaxation of the papilla and to the flow of bile. The magnesium sulphate remains in the duodenum only a few seconds; and while it may have a primary effect, as other things would, it is hard to see how this would be more than a temporary effect; as, after a few seconds, the solution has passed out of the duodenum and no longer exerts any influence.

The method described by Dr. Einhorn of stimulating bile flow by a cup of bouillon or tea may prove as useful as the introduction of chemicals.

The question whether turbid bile is a correct indication for operation is an important one. Personally, I am one who believes that many of these cases are cured spontaneously or under treatment, and I think we are going too far in considering turbid bile of and by itself an indication for operation. Dr. Meyer has shown that the turbid bile is sometimes sterile. Even bile infected with streptococci does not of necessity indicate an infectious disease of the gall-bladder. The indications will always remain a clinical problem. Certain it is that the bacteriology of the bile obtained by Lyon's method may prove to be one important addition to the evidence to be considered.

Dr. Joseph Sailer, Philadelphia: Dr. Bettmann has made such an excellent discussion on Dr. Lyon's paper, as to leave little for me to say.

I can, of course, begin with the statement that Dr. Einhorn's method and others' introduced lately are diagnostic of gall-bladder conditions and conditions of the upper portion of the abdomen. If I might speak of Dr. Lyon's method, which I have used a few times, it is proper for me to bear testimony to the fact that it works; that it is a practical method, and fairly large quantities of bile can be obtained by the procedure he describes. Therefore, we have something definite on which to work. We begin by having in our hands a large amount of the secretion of not only the gall-bladder, but also the bile ducts and the pancreas, and possibly in some cases we can distinguish between the bile from the different portions. I have not had the same success in that respect as Dr. Lyon has had, possibly due to deficiency in technique on my part. This is merely a criticism of myself. I think also, however, that this method is going to be considerably simplified before it enters into general use. At present it is an extremely complicated one and this is a great disadvantage at the present time. It is a form of experiment, and every precaution that can be placed around an experimental investigation to minimize error is important and should be employed. In all clinical methods, this has been the case. Some of the precautions are, perhaps, unnecessary and can be omitted later, but Dr. Lyon in using as many precautions as he could devise, has added greatly to the usefulness of the method.

The third feature that I want to emphasize, I hope, again will not be regarded in any way in the light of a criticism; but the conclusions of Dr. Lyon regarding the different portions from which the bile is derived are extremely plausible, although at present scarcely more than plausible. They are not definite. The experiment has not broken down, but it is not yet definitely conclusive. So before we accept unreservedly these conclusions regarding the source of different forms of bile and different factors in the method, we must wait for a little more light. Therefore, we should not now regard this method as giving us the final results. It may, in time, give these results; but if it does, it will be almost unique; because one of the great errors that we make in the utilization of various laboratory methods is to expect that their results are going to be absolutely definite. There are very few tests whose results can at present be regarded as absolutely definite. They are contributory adjuncts to the making of a diagnosis.

Now there is still another thing that I wish to speak of. There are three points about the use of this method that I should like to emphasize. One of these Dr. Lyon has already touched upon. There has been, of late years, an enormous amount of theoretical work done on focal infection. I used the word "theoretical," I think, advisedly, because if anyone for a moment considers the enormous number of teeth that have been sacrificed on the altar of the God of Focal Infection without any particular benefit to the patient, he must recognize that a collection of pus somewhere in the body does not necessarily mean that this collection is responsible for all the disabilities from which the patient suffers. Sometimes we find the important collection of pus, but not always. It is important if we find an abscess of the root of a tooth, to see whether there may not be a collection of bacteria in some other portion of the body. We might find out whether it is in the appendix, by removing the appendix. That is a simple operation at the present day, and one which surgeons enthusiastically perform. We cannot, however, open the abdomen and remove the gall-bladder with such indifference. It is a more serious operation, and if it can be shown that Dr. Lyon's method gives information regarding the condition of the bile ducts, the gall-bladder or the liver itself, it will be an important addition to our diagnostic methods.

I think, also, that there is going to be great opportunity in this method for the diagnosis of splenic anemia, and probably, likewise, in the determination of deficiency in the secretion of the pancreas.

Dr. Jacob Kauffman, New York City: There was scarcely any need for this discussion to Dr. Lyon's paper to prove what we may expect from a further elaboration of the examination of duodenal contents, in the diagnosis of diseases of the biliary tract. I advisedly say biliary tract; for I think that we should not differentiate too sharply between disease of the bile ducts and disease of the gall-bladder. Almost invariably, where you have disease of the gall-bladder, you also find disease of the bile ducts, the large and the small ones. In a most interesting article on "Jaundice and Its Relation to Cholangitis and Cholangiolitis," published last year, Naunyn in his masterful way brings forth convincing arguments for his contention that diseases of the biliary tract are the result of inflammatory processes of varying degree and that as a rule the biliary tract as a whole is involved. It is important to keep that in mind, because it explains the frequent recurrence of disturbances and of inflammatory processes after the removal of the gall-bladder; with the gall-bladder removed you still have the original process in the bile ducts. That leads us to the discussion of the indication for removing the gall-bladder in those cases which Dr. Meyer referred to and for which, in a rather general way, he advocates cholecystectomy. I think that it is perfectly legitimate to remove the gall-bladder when inflammatory processes of a more severe type have led to such destruction that the function of the gall-bladder is abolished and it has become useless. That, however, is not the case in all these patients, and not even in the majority; and it is important to realize that, according to recent investigations, the gall-bladder has a more important function than merely to act as a reservoir for bile during the interdigestive period. It has been shown that gall-bladder removal in animals reduces the digestive secretion of the stomach and of the pancreas, which indicates that the activity of the gall-bladder plays a rôle in the elaboration of digestive secretions. That is corroborated by clinical findings. We know that at the beginning and during a certain part of the course of gall-stone disease we often find high values for gastric secretion, that after a protracted course and particularly after a number of attacks of severe cholecystitis we find a decrease of gastric secretion, and often a complete achylia. Favrel, of Richmond, in a recent paper, suggests that this experience be used by examining the stomach contents in all cases of gall-bladder disease, to see whether the gall-bladder is still functioning. If that is the case, I think that we should ponder before we remove the gall-bladder. The inflammation of the gall-bladder and the bile ducts are not always of such a character that they cannot be remedied by medical treatment. We have to look for causative factors first.

Dr. Lyon mentioned one type, the so-called migraine type, which is liable to lead to disease of the bile duct. That is perfectly correct. I shall not go into the working of this process, but I should like to mention another type that I frequently see; and that is the type of high livers, people who are accustomed to eat and drink a lot of rich, heavy food without getting the proper amount of exercise. You find them with heavy, bloated abdomens, and almost invariably with large, palpable, swollen and tender livers. This condition of the liver indicates the state of congestion that exists in the upper abdomen, the liver and the bile ducts. If, for one reason or another, this congestion gets aggravated, then a dormant inflammatory process becomes acute, and that leads to these acute attacks. We are here confronted with the same situation as in what we call "catching cold." It has been shown that when you rapidly cool off an animal, the mucous membrane of the pharynx becomes congested and thereby more susceptible to the invasion of bacteria, normally inhabiting the throat. A similar situation exists in these cases of plethoric high livers with congested bile ducts, when an aggravation of the congestion brings forth an active invasion of bacteria, otherwise innocuous. Proper dieting in connection with various methods of medical treatment are often successful in reducing congestion and with that of recurrent attacks of inflammation.

Dr. Alexius McGlannan, Baltimore: The surgeons are divided into two classes: Those who look upon cholecystectomy as an operation of choice; and the minority, who are not of this opinion. As one of this minority, it is gratifying to hear of surgeons who are not of this opinion in regard to gall-bladder lesions. One of the principle arguments of those who favor cholecystectomy has been the presence of the streptococcus in the wall of the gall-bladder. No one would recommend the amputation of the hand on account of such an infection of the bone, or tendon sheath, or removal of the urinary bladder for chronic cystitis. Both these operations can be done with a low mortality, however. That is not a reason for doing them. That the gall-bladder has a function, we must admit. Those who have had to open the abdomen after cholecystectomy have found the stump dilated, and the portion of the cystic duct remaining dilated, evidently an attempt on the part of the body to restore whatever function is associated with this portion of the bile apparatus by this dilatation.

Those who have looked over the literature since cholecystectomy has been so widely advocated must be struck with the number of plastic operations described for the restoration of the bile duct. Why should this be necessary? The paper of Dr. Lyon has brought out the striking fact that there is disease of the bile duct saparate from, as well as associated with disease of the gall-bladder. Removal of the gall-bladder in the presence of diseased bile ducts cannot cure the patient. We hope to reach a point of differentiation between disease of the gall-bladder or bile duct alone and cases in which the two are associated. I hope that the pendulum will swing, and that we shall be looked on as conservative surgeons.

Dr. John C. Hemmeter, Baltimore: At no time in the past generation has there been a period in which there have been such important and critical development in the understanding of cholelithiasis and biliary disease in general as at the present time; and two of the foremost clinicians the world has ever seen will shortly come out with new works on the subject, namely Naunyn and Wenkebach, in Vienna, whose associates are working with duodenal intubation for the diagnosis of gall-stones.

I was the first clinician to devise a method for intubating the duodenum in 1896. I have practiced duodenal intubation for twenty years, and in none of these, except for about two months in each year, was there a time that I did not have a case on which duodenal intubation was not practiced. I think Dr. Lyon's method is not only feasible, but is the most promising that I can think of. There is no reason why we should not use Dr. Einhorn's method, but Dr. Lyon's results should be compared with those obtained by it. However, there is really one objection. By adding magnesium sulphate, a chemical is added that leads to confusion in the quantitative results of subsequent analysis. Dr. Lyon, in one of his papers, makes the observation that I have made. He states that he has had cases to return to him after removal of the gall-bladder. I have had two operations in three years, in one and the same case in which the gall-bladder was removed.

Dr. Julius Friedenwald, Baltimore: I am quite in accord with Dr. Einhorn and Dr. Meyer regarding the diagnosis of chronic cholecystitis. The direct examination of the bile gives an additional evidence of great value as to the character of the disturbance. Dr. Lyon's paper is also of much interest. I have been utilizing Meltzer's method of evacuating the bile into the duodenum by means of an instillation of magnesium sulphate solution, and aspirating the contents according to Lyon's method. This method of examination, and the treatment by means of duodenal lavage with sulphate of soda and magnesium sulphate according to Aaron's plan or with Ringer's solution or a solution of permanganate of potash, has been of great value in a number of my cases.

Dr. Einhorn (closing): With regard to the method of Dr. Lyon or the necessity of using magnesium sulphate, I must say that I personally do not find it necessary. I usually examine these patients if I want to see the bile in a fasting condition; and if I do not get any bile by aspiration, then I instil saline or plain water at about 100° temperature about 5 to 10 c.c., let it run in and come back. That is usually followed by bile—first a little that is discarded, and later by more; so that we are able to get a specimen for what we want. In nine cases out of ten we get specimens enough without anything. I think that is the best way.

Regarding the quantity of flow, I try to estimate that, too. By aspiration alone, that is not sufficient; but after the aspiration has been started, we let the barrel of the syringe drop below the level of the duodenum, and let it rest in that position for five or ten minutes, to see how much comes out drop by drop. In most cases, we can estimate how much that is. In hypersecretion, we cannot tell usually whether bile alone or pancreatic juice plays the greatest rôle. What is of importance is that while in most instances, in normal individuals or those in whom there is not much disease around the gall-bladder, we usually get clear bile, in those diseased we find abnormal conditions. Gastric juice alone cannot do this, because we have to deal with patients in whom the stomach is usually empty—fasting conditions.

As regards surgery, I did not recommend it in each case of cholecystitis. I said that when the attacks are very frequent, when they interfere with the mode of life of the patient; when he have to deal with chronic conditions that debilitate the patient and we run the risk of losing the patient, I recommend operation; also in the case of hospital patients who are near death, and would probably die if left alone. Operation in these cases must be done. If we do it and find no stone, but find dirty bile, this is an indication to remove the gall-bladder; that is what I said.

Dr. Meyer (closing): These investigations are only just beginning. We have added to the examination of the duodenal contents this examination of the bile during operation, and have tried to build on our findings the indication for operation. To my mind the future only can show whether patients, who have exhibited on pathological examinations bacteria in the wall of the gall-bladder and whose gall-bladders are chronically diseased, can really be cured by medical means. If so, we will certainly stop removing gall-bladders.

We ought to know, and all should join in the attempt to find out definitely, whether in the normal human being the color of the bile is a golden yellow and whether turbid bile is a symptom of disease. It would be very nice if we could get permission from every patient in whom we open the abdomen, to aspirate the gall-bladder. I believe that by using a fine needle and penetrating the wall obliquely we would not do any harm. But where is the patient who would allow it? Nowhere.

We also operated on patients without the presence of fever. I do not want it to be thought that we only operated when fever was present. We always operated on patients with turbid bile, if the symptoms pointed to trouble in the upper abdomen, and examination of stomach and duodenum was negative. It is our intention to collect the final results.

The important point is that the examination of the duodenal contents should be done systematically in every patient suspicious of having gall-bladder affection. So far it is not done and was not done. We had the X-ray and the cholesterol test; now we should add in these cases examination of the duodenal contents in every hospital where there is a laboratory.

Dr. Lyon (closing): It was impossible for me to present what I had to present in a few moments and my most important purpose was to try to give you a review of methods of earlier diagnosis so that we may learn how to catch our gall-bladder cases before pathological lesions have been formed, and thus cut down the incidence of the later and more extreme phases of gall-bladder pathology which, naturally, are the points of correction that the surgeon has had to concern himself with in the past. If the surgeons could get their cases earlier, so that the associated pathological lesions in the upper right quadrant had not become so well established, their results would be better than they are today. There was not sufficient time to present adequately the possibilities of treatment by this new method, but thus far our results have been most encouraging.

In answer to Dr. Bettman's question about nervous conditions producing an excessive flow of bile, I would say that I believe this is correct. Nervous conditions cannot, however, alter the bile and make it pathological. Increased bile is a different thing from abnormal bile. Nervous conditions produced by train and ocean trips may mean that the patient had a disorder that we have not yet learned to recognize. The question of food chemistry being an important factor in the discharge of bile is a part of the process, but not all. Another fact we should remember is that sphincter relaxation of the common duct does not mean that the gall-bladder necessarily contracts and discharges its bile. There are meals (starches and carbohydrates) that require a greater amount of pancreatic juice. This is delivered through the relaxed duct sphincter, but the gall-bladder does not so well empty with this type of food chemistry. Peptone and albumose meals are a better stimulus for contraction of the gall-bladder.

The question of high livers is important; but the question of low livers is just as important. If it is the food chemistry that causes the discharge of bile, then going from breakfast to dinner without this stimulus predisposes as much to biliary stasis as does high living.

One other fact that I want to bring out is that every chemical substance does not cause the sphincter to relax. Certain things have a selective affinity to make the gall-bladder contract, and other things have a selective affinity to make the pancreas discharge its juices. Benzol benzoate relaxes the sphincter, but does not make the gall-bladder contract; while magnesium sulphate does, and has a relation to the process to which Meltzer called attention. Of course, we cannot be sure where the B bile comes from; but logical deductions are reasonable. If you have so much pathological static bile stored up in the gall-bladder and a closed duct sphincter, and you relax the sphincter with magnesium sulphate, and recovering a lot of tarry bile, preceded and followed by other types of bile, where is it coming from? It is not from the liver, except in those cases with biliary cirrhosis. Rous is going to show, at the College of Physicians, that the gall-bladder possesses the power to concentrate the liver bile. Therefore this static, more concentrated bile is presumably coming from the gall-bladder. If you find high columnar bile-stained epithelium of the gall-bladder different from the low columnar type of epithelium from the ducts, you can match up your cytological impression with the gross appearance of the bile. The method will not be popular, because it is too time-consuming and laborious; but with it we can improve our methods of diagnosis of gall-bladder disease and can open up a new field of treatment. Thus men like you, who will do things that require three hours to do and not half an hour, will get the information where the lazy or indifferent man will not.

Dr. Sailer says that he did not get similar results, probably, as he says, because he did not take the time. You must match up your bacteriology and put it through selective animal affinity tests, if it is going to be done really exactly.

Dr. Kauffman said that the gall-bladder has a function. He is right. But is it functioning? Is it capable of discharging its contents if assisted? If so, it has not completely lost its function.

Dr. Hemmeter spoke of magnesium sulphate confusing the chemistry. That is true, if not removed promptly. It is surprising how quickly bile will be recovered after magnesium sulphate is introduced. Then comes the question, how soon is the gall-bladder bile discharged; and this gives an inkling as to how much pressure it is under, waiting for a stimulus to contract. Magnesium sulphate stays so short a time in the duodenum and your later supply of pure bile is so large in amount, that I do not believe that the chemistry is going to be materially altered.

THE ROENTGENOLOGIC DIAGNOSIS OF GALL-BLADDER LESIONS.*

By Dudley Roberts, M. D.,

NEW YORK.

It is the common belief that only about 10 or 15 per cent. of gall stones can be visualized, and that the demonstration of the pathologic gall-bladder is largely chimerical. This belief is based on the character of roentgenographic work that was possible several years ago, and is untenable at the present time in view of the advances that have been made in technic which bring out "soft part detail." It is true that the making and interpreting of gall-bladder roentgenograms is an extremely laborious undertaking, but the importance of the subject can hardly be exaggerated, in view of the frequency of diseases of the gall-bladder and the uncertainty of their clinical diagnosis in a large proportion of cases.

The present pessimism arises from the mistaken notion that the lime content of the majority of stones is so small that their visualization is absolutely impossible. It is not generally appreciated that few gall stones are devoid of a lime-containing layer, and even these few may be shown, given adequate detail, because they are less dense than the surrounding tissues or the fluid in which they lie and hence will cast negative shadows. There are at present insuperable obstacles to the visualization of all gall stones, but as the technic is developed and simplified the Roentgen diagnosis of gall stones will be put on a thoroughly practical and reliable basis, with only a negligible proportion of unavoidable errors. The enlarged gall-bladder can easily be visualized, but at present it seems that there will be a small residue of cases of chronic cholecystitis without stones or dilation which cannot be determined by roentgenography. In view of the large percentage of known stones which we have been able to visualize, the importance of negative findings cannot be lightly dismissed. One cannot properly estimate the importance of negative findings on the basis of the percentage of failures in a demonstrated series because the failures occur in heavy subjects. The importance to be attached to negative findings depends on the character of the roentgenograms secured. In some cases the results do not warrant a negative opinion; in others the results may be fairly satisfactory and make it improbable that stones are present; but the perfectly satisfactory series, that is, an adequate number of roentgenograms showing a wealth of detail, may warrant a negative diagnosis of stones or enlarged gall-bladder.

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*Owing to the impracticability of reproducing detailed roentgenograms of the gall-bladder region satisfactorily, illustrations have been omitted. Reprints will be furnished by the author on request, accompanied with a set of illustrative reductions.

Satisfactory Gall-Bladder Roentgenograms.

The satisfactory roentgenogram for gall stone diagnosis shows sharp, clear-cut lines of the hepatic flexure which indicate perfect stillness and in addition a detail of soft structure that is difficult to describe. Certainly the entire outline of kidney should be very clear, even the indentation of the hilum being obvious. The liver edge is always very evident, and serves as a guide in the search for the gall-bladder. Each exposure covers a small field, but the entire area from well above the ninth rib cartilage to the crest of the ileum must be roentgenographed. The plate or film darkened by "body rays" is lacking in contrast, as seen particularly by the failure of the lowermost ribs to give bone detail.

Exposure Technic.

Three closely interrelated problems present themselves in gall-bladder roentgenography: how to prevent motion during the exposure, how to avoid overpenetration, with consequent loss of fine gradations of density, and how to prevent the blurring of the roentgenogram with rays other than those which arise at the focal point of the target.

Absolute stillness during the exposure is essential for the visualization of all except about 20 per cent. of stones, as the density of the shadow in the path of the rays decreases directly with the extent of its excursion during the exposure. The stone with a calcium layer only one thirty-second inch in thickness casts a perfect and unmistakable ring shadow if it is perfectly stationary, but will be half as dense if it moves one thirty-second inch. The patient must be watched and drilled in sustaining forced expiration during the exposure, and the results must be carefully studied to make sure that not the slightest movement is shown. Furthermore, the exposure must be as short as is consistent with satisfactory work. The shorter the exposure the less chance of motion. Some subjects must be roentgenographed in an extremely short time regardless of the loss of detail, because they cannot cease respiration for more than a fraction of a second. Overpenetration and lack of detail results from speed. Fine gradations of density can be secured only by the least possible penetration, sufficient time being used to give the desired density of the roentgenogram. Duplitized films and fast double screens have made possible the use of rays of low penetrating power, which was entirely impossible with unscreened plates that required many times the exposure. The use of duplitized films and two intensifying screens we regard as the most important advance of recent years; and as still more sensitive screens are developed, gall-bladder roentgenography will be made more uniformly successful.

The exclusion of "aberrant" rays from the tube is accomplished best by the use of a small lead diaphragm, a long cylindric cone and a filter of 0.5 mm. of aluminum. The best results have been secured with a fine focus hydrogen tube. The exclusion of rays that are generated in the body of the patient has not yet reached a practical stage, and we must depend on lower voltage currents, which are less prone to generate such rays even though the time of exposure in heavy subjects is so long that many exposures are rendered useless by slight respiratory movements.

Our most satisfactory results are secured with a fine focus gas or hydrogen tube, a plate target distance of from 28 to 30 inches, a small diaphragm and long cone giving an exposure field of only five or six inches. A parallel spark gap of two and one-half to three and one-half inches is regularly used, and a milliamperage of from 20 to 30 in the tube. Exposures are made with the patient prone on the cassette during forced, sustained expiration. The effort is made to underexpose the film slightly and push the development short of causing fog or stain in the effort to bring out detail.

The Interpretation of Gall-Bladder Roentgenograms.

With increasing detail the percentage of stones that are definite at a glance is greatly increased, but the majority of stone diagnoses are safely reached only by prolonged study and comparison of different exposures. The reducing camera has come into routine use in the study of all doubtful cases, as the results it gives in the concentration of images are extremely valuable.

Indirect suggestive evidence of a gall-bladder lesion is frequently given by the study of the stomach, duodenum and hepatic flexure of the colon at different intervals after the ingestion of an opaque meal. Antrum and cap distortion not characteristic of ulcer may be extremely suggestive of gall-bladder adhesions, and the constant concavity on the inner side or above the hepatic flexure may serve to locate a dilated gall-bladder which subsequently is intensively studied. The greatest care must be exercised in drawing conclusions from these apparent distortions. On the whole, we feel that the chief reliance must be placed in direct evidence.

The recognition of gall-bladder lesions from the roentgen standpoint depends on either the visualization of the gall-bladder or the demonstration of stones or both.

Gall-Bladder Shadows.—From clinical and experimental studies of removed gall-bladders and bags containing various solutions and specimens of bile we have reached certain conclusions that have an important bearing on interpretation of gall-bladder roentgenograms:

1. A bag of fluid in the upper abdomen can be visualized more or less distinctly, according to its anteroposterior thickness or mass.

2. The density of the image of such a fluid bag varies imperceptibly with fluid. One might expect that differences in specific gravity would result in differences of Roentgen-ray density, and this would be true were the mass sufficiently great. We have been unable to discover that any pathologic bile casts a shadow that is denser than an equal mass of water or normal bile. It is in our opinion futile to talk of a pathologic gall-bladder casting a recognizable shadow because it contains abnormal bile.

3. Pathologic tissue, such as new growth or a thickened gall-bladder wall, may indeed be visualized as is the kidney if its mass is sufficiently great, but it is a rare gall-bladder wall that is sufficiently thickened by inflammation to give rise to a definitely recognizable image. Its density is no greater than the walls of the surrounding hollow viscera, which in themselves are certainly

not demonstrable. New growths of the stomach are only visualized indirectly by the effect they cause on the opaque cast of the hollow viscus.

4. The visualized gall-bladder is one sufficiently dilated and increased above the normal in its mass to give a definite image. The contention of George that the visualized gall-bladder is a pathologic gall-bladder seems in general to be correct, although it is extremely probable that the normal gall-bladder will sometimes be so placed that its visualization will be definite. Just as at operation the size of the organ is a criterion of the condition of the organ, so from the roentgen aspect we must be prepared to express an opinion that a gall-bladder is or is not pathologic on the basis of its apparent size.

5. Fluid in general is more dense than gall stones with a low content of lime salts. Experimentally we find that stones immersed in fluid and roentgenographed cast a positive or a negative shadow or no shadow at all, according to the density of the stones and the thickness of the layer of fluid. When a removed gall-bladder is roentgenographed, stones show if there is little fluid, regardless of their composition; and if fluid is injected into the bladder until it is actually dilated, the stones tend to disappear as the fluid is increased. Evidently not all stones can be shown in the living subject, because they lie in too much fluid. Stones may show in the neck of the bladder and not show at all in the fundus where the fluid mass is greater.

Characteristics of Gall-Bladder Shadows.—In view of the fact that fluid in a gall-bladder may obscure stones, and a dilated gall-bladder without stones is fully as important as stones, it is necessary that the roentgenographic appearance of the gall-bladder be carefully studied.

The gall-bladder is sharp in its outline and suggests a flaccid bag. Normally it lies in a broad hollow on the under surface of the liver, the fundus reaching about to the liver edge. From the fundus it extends upward, backward and inward. Usually it is within an inch of the right side of the spine. The variations from this average course and location of the bladder are many. Sometimes its long diameter is across the abdomen, the fundus reaching almost the extreme right side of the liver. In the enteroptotic type it is normally vertical because the front part of the under surface of the liver is vertical. Normally it is plastered on the under surface of the liver; but with a gradual increase in the amount of its contents it tends to become sessile. Depending on the manner in which it is bound down, either the fundus is elongated or the long lower surface bulges downward. In an anteroposterior roentgenogram the image of the gall-bladder will be determined by its size and extension. It may be caught on its long axis, giving rise to a round, dense area, or it may be shown in its entire pear-shaped profile. Accurate gall-bladder recognition is extremely difficult, and the only safe interpretation is to exclude as gall-bladder shadows all suggestive dense areas in this region which have not the peculiarly definite, sharp rounded contour. The sharply visualized kidney is easily mistaken for a gall-bladder, particularly when it lies almost transversely below the liver, as is frequently the case in women. The greatest difficulty is encountered in the study of those roentgenograms in which the shadows of liver, right kidney and gall-bladder are superimposed. The dilated gall-bladder also may cast a shadow through the liver tissue which overlies it, and the combination must be worked out by prolonged study.

Variety of Stones and Their Roentgenographic Characteristics.—A knowledge of the principal varieties of stones and the kind of shadows they cast is a great help in interpretation. In the subjoined classification of stones, which is a combination of the descriptions given by Naunyn and the later one of Aschoff and Bacmeister, we have connected the roentgenographic findings with the chemical and structural peculiarities of each class.

1. The Radial Cholesterin Stone: This rare type of single stone occurs in gall-bladders that show evidence of dilatation without inflammation. Its structure is peculiar, in that it is composed of pure cholesterin crystals that radiate from the center to the surface. This stone is less dense than all surrounding tissue and can be visualized only as a negative shadow, that is, a round or oval dark spot.

2. The Combination Stone: When inflammation develops in a gall-bladder that contains a radial cholesterin stone, the inflammatory exudate causes a deposit of lime salts on the stone, and this layer shows as a ring or oval shadow. It sometimes happens that the lime is unequally deposited and only a segment is visualized.

3. Cholesterin-Bilirubin Calcium Stones: This rather common

type is fairly large, from two to five in number, frequently faceted and nested in the neck of the gall-bladder or the cystic duct. They are usually not layered, and the calcium salts are equally distributed through the mass of the stones so that they do not show as rings or triangles, but as solid spots or an elongated finger-like dense area due to the nesting of the stones. The percentage of lime is very small, and the shadows are lost by the slightest movement or overpenetration or darkening by "body rays."

4. Common Multiple Faceted Stones: As this group represents nearly half of all stones, familiarity with their size, structures and roentgenographic characteristics is most important. Their number is usually great, seldom less than twenty, frequently several hundred. They are usually small, faceted and very irregular in shape. They are composed of cholesterin and bile salts with very little intermixed lime, but with a thin coating on some facets of some of the stones in a considerable percentage of collections. The visualization of some of these stones is exceedingly easy because of the dense lime coating, which gives rise to an irregular mosaic. The greatest detail is necessary to visualize the majority, and one must learn to recognize the faint mosaic of the gall-bladder filled with such stones. This mosaic or spotted appearance is due either to the slight coating of stones with lime salts or to negative shadows made by the readily penetrated stones outlined by the denser bile. When the gall-bladder is crowded with relatively soft stones, the mass is sometimes sufficient to cast a definite faint shadow of a gall-bladder in which no individual shadows can be seen. These negative shadows are hard to interpret as stones because their outline is diffuse. No matter how great detail we are ultimately able to obtain in this work, fully 15 per cent. of stone diagnoses must be made by recognition of this persistent spotty appearance made by these soft stones in the fluid gall-bladder contents. At present it is impossible to secure evidence of this type of stones in heavy subjects, and allowance must always be made for this fact in making negative diagnoses.

5. Pure Bilirubin Lime Stones: These occur either as small seed-sized, soft black granules, or as several larger stones. While they are extremely soft and friable, they contain a definite ad-

mixture of lime, and only their small size makes their recognition impossible.

6. Calcium Bilirubinate Stone: This stone is usually single and of flinty hardness throughout, and consequently it gives rise to solid, unmistakable shadows. It unfortunately represents only about 5 per cent. of all gall stones.

Case Series.

Statistical studies of diagnostic results are extremely unsatisfactory, as chance enters so largely, particularly if the series of cases is small. Negative findings and corpulence tend to postpone confirmation of diagnosis by exploration, and total percentages of results are hard to reach. Two striking facts, however, are evident from a survey of our experience of the last year and a half, during which the present technic has been a routine. First, in a "known series" all stones have eventually been visualized. Second, while several times we have been led into stating that there were probably stones in cases in which no stones were subsequently found, we have not failed to find stones in any case that was studied for stones when subsequent operation demonstrated that stones were present. Nevertheless, this true statement is misleading, as in three cases in which operation has not yet been performed, stone shadows could not be found because of the size of the patient and consequent unsatisfactory results, although we feel certain that they are present.

Series 1.—*Known Stone Cases.*—At the Woman's Hospital, New York, in 18 patients in whom stones had been found and not removed because of the length of the pelvic operation, our subsequent roentgenographic study has eventually demonstrated the stones in each case as shown on the operative notes. In this series three were extremely easy to visualize and three extremely hard, requiring several attempts to secure adequate detail. By chance alone the heavy patients had fairly dense stones, or the results would not have been 100 per cent. Eventually this series will be large enough to determine the percentage of stones that can be visualized when of a size sufficient to be felt through a lower abdominal incision.

Series 2.—*Suspected Stone Cases.*—In a recent consecutive series of 70 cases intensively studied because of a definite clinical history of gall-bladder disease, the Roentgen findings have been positive in 45 and negative in 25 cases. Thirty of the positive and 19 of the negative cases have been explored. Three of the positive cases were incorrect, two mistakes being due to the accidental use of the same defective screen in everyone of a short series of exposures, and the third showing a shadow due to calcification of undetermined site outside the gall-bladder. No stones were found in any of the negative cases, but three on removal showed chronic catarrhal cholecystitis without dilation. No stones could be visualized in three cases in which operation had not been performed, in which there certainly seemed to be stones.

Series 3.—In the Woman's Hospital Clinic it has repeatedly happened that young mothers have been referred for a diagnosis of upper abdominal complaints that seem very definitely to be gall-bladder inflammations without stones. The group has been an exceedingly definite one, and interesting in that we have been unable to visualize the gall-bladder. This experience in such a large series of cases has convinced us that the catarrhal unobstructed gall-bladder is not demonstrable, and that the normal sized gall-bladder can rarely be demonstrated. For early diagnosis of gall-bladder inflammation we must rely on the clinical picture and possibly the examination of extracted bile, as suggested by Einhorn.

Conclusions.

1. The roentgenologic diagnosis of gall-stones and the dilated gall-bladder with a small percentage of failures is possible at the present time.

2. Negative diagnosis has a value that is proportionate to the intensity of detail and sharpness of image secured in the given case. Negative diagnosis has very little value in subjects so heavy that satisfactory roentgenograms cannot be made. Subjects of slight or medium body thickness can be roentgenographed with an intensity of detail that justifies an experienced interpreter in a negative diagnosis of stones or a dilated gall-bladder.

3. The roentgenologic diagnosis of gall stones necessitates such an expenditure of time and money in the taking of satisfactory exposures, and so much experience in the interpretation of intensely detailed roentgenograms, that it is not at present a safe and practical method of diagnosis for general adoption. Ten or 15 per cent. of stones can be plainly visualized even by an

extremely poor equipment and technic; but under such conditions the importance to be attached to negative findings is negligible.

4. Gall-bladder roentgenograms of satisfactory detail can be made only with direct rays of low penetrating power, and duplitized films with fast screens.

5. An insuperable limitation of roentgenologic diagnosis of gall-bladder lesions is the apparent impossibility of securing roentgenographic evidence of chronic cholecystitis without dilation or of new growth of the gall-bladder and biliary ducts.

DISCUSSION.

Dr. Lewis Gregory Cole, New York City: In discussing this question of X-ray diagnosis of gall-stones and gall-bladder pathology, I have alternated from the optimistic to the pessimistic side from time to time. When I am optimistic, I work as hard as George and Roberts; when I am pessimistic, I know I do not. I have had a streak of optimism in the last five weeks and have worked with all the energy and zeal, and what skill I may possess, to get the detail in gall-bladder plates that I considered satisfactory. I have no doubt that others may say that these plates are not satisfactory, but at least they were the best that I could get.

Instead of getting closer to the solution, however, it seems to me that I am getting further away because, in a large percentage of these plates, I was able to show such detail that the spine, tenth and eleventh ribs, transverse processes, psoas muscle and the outline of the kidney could all be seen, and, in some plates, one could actually visualize the descending duodenum without barium having been administered. In many of the plates I found a shadow that I think was sufficiently clear to be detected by a great majority of observers with no more than a moderate amount of imagination—a shadow which represented the gall-bladder—and yet I am not at all convinced that the shadow that I saw was a pathological gall-bladder.

I see no real obstacle to prevent a normal, bile-filled gall-bladder from being shown, and, frankly, I have not known how to interpret these plates. I have stated in my reports that the gall-bladder was visualized. Dr. Steiner, my associate, said: "That is not fair, because anyone who gets such a report will assume that it is a pathological gall-bladder." I do not know whether this is true or not. A number of these gall-bladders have been opened and taken out, several by Dr. Erdmann, who recently said: "You can tell those people that I am through taking out normal gall-bladders on X-ray findings."

I do not know just where I stand today, and I had a great deal of uncertainty as to whether I should indulge in the discussion of Dr. Roberts' talk this morning or even of Dr. George's tonight. I hope, after the session has adjourned, that we may get together and look over these plates which we have recently made. I have done 65 cases since I had this spasm of increased energy, and I have them all here. Some are satisfactory and some are not. I have grouped them by taking 30 cases that were fairly satisfactory, and I am not saying how many proved to have gall-stones and how many did not. Perhaps we can get together this afternoon and look over these plates and perhaps we can do something else which I have wanted to do before, and that is to take ten gall-bladder plates and send them to George with the following questionnaire: (1) Do you consider these satisfactory plates on which to make a diagnosis? Answer yes or no. (2) If they are satisfactory plates on which to make a diagnosis, do you believe that there are stones in this case or do you not? (3) Do you believe that the gall-bladder is pathological or do you believe that it is not?

I should then like to take the same plates and send them to Roberts, Mills and any others who think they can diagnose gall-bladder disease, with the same questionnaire. I believe that the answers from such a query would go far toward settling the present differences of opinion that exist.

I realize that some of these men will say that the plates are unsatisfactory, but I flatter myself that at least ten of this series which I have selected are quite as good as the plates in routine procedure on which such diagnoses must be based.

I am not down on the program to discuss Dr. George's paper tonight, but perhaps after he has finished reading it I may have something to say.

Regarding the negative and positive shadows of gall stones, I believe that what Dr. Roberts saw was a gall-bladder filled with stones and with a relatively small amount of bile. The stones show as negative shadows and, with a small quantity of bile between them, develop a mottled or ring-like appearance, and the reason for their disappearance is that with an increased quantity of bile the stones become submerged, the negative shadows are obliterated and, as a consequence, the ring-like appearance disappears. This can be demonstrated by submerging removed stones in water and then making plates.

BLOOD CHOLESTEROL IN GASTRO-ENTEROLOGIC CASES.

By Dr. Truman G. Schnabel,

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Cholesterol or cholesterin, a monatomic alcohol, is found as such in the human blood stream. In combination with fatty acids it forms, among other esters, cholesterol palmitate and cholesterol oleate. A fairly definite relation or balance is maintained between the free and combined cholesterol of the blood. Cholesterin is also found in the nervous system, bile, milk and other body tissues and fluids. There is some uncertainty as to the exact chemical structure of cholesterol.[1] It is soluble in ether, chloroform, benzene, fats and hot alcohol.

The origin of cholesterol is, for the most part, exogenous, although there is some evidence that it may have an endogenous source.[2] It is readily absorbed from the intestinal tract contents through the chyle and may be recognized free or esterized in the contents of the thoracic duct.[3] The blood cholesterol content can be readily increased by feeding foods rich in this substance. That the biliary cholesterol can be increased in like manner remains uncertain. Feeding experiments leave this point in doubt. The origin of biliary cholesterol is, perhaps, to be found in the disintegrating blood cells of the body. Bile is the only suitable solvent in the body for cholesterol, but whether the bile is a normal mode of excretion for this substance is just as uncertain as other facts pertaining to this alcohol. Fistula bile is poorer in cholesterol than gall-bladder bile, and some, therefore, believe that this substance somehow or other has its origin in the lining membrane of this organ. Some contend that this difference in the bile from these two sources soon ends after the unusual conditions imposed by the operation cease[4]; recently, Rous and McMaster[5] seem to have demonstrated an ability on the part of the gall-bladder to withdraw normal salt solution from the hepatic bile and thereby concentrate the latter fluid. There is no reason to believe that cholesterol is changed to cholic acid, a substance participating in the formation of bile salts. The liver probably is the regulator of cholesterol metabolism and maintains the level by excretion through the bile.[6]

Both lecithin and cholesterol assume some role in the transportation and metabolism of fats. The esters of cholesterol are found in the red cells and are practically absent during fasting. The presence of this substance in the red cells probably accounts for the role the latter play in fat metabolism.

The amount of cholesterol normally found in the blood has been variously stated. Bacmeister and Havers[7] give 110 to 180 mg. per 100 c.c., and Widal, Weill and Laudat[8] 174 to 195 mg. per 100 c.c. Bloor[9] found the average value for men to be 210 mg. and 230 mg. for women. A wide variety of normals are given by other workers. It is probably fair to say, however, that an upper normal is about 200 mg. per 100 c.c. of blood, while the lower level is about 150 mg.

The literature is in accord on the findings of a low blood cholesterol in pernicious anemia, which probably has its basis in the diminution of the corpuscular elements to be found in this disease. To beriberi, syphilis, prostration, cachexia and fever are accredited low cholesterol findings, and recently a low blood cholesterol[10] has been found to be of bad prognostic import in nephritis. In nongrave nephritis, arteriosclerosis, obstructive jaundice, diabetes and pregnancy high figures are said to prevail. The literature is by no means in accord as to the constancy of blood cholesterol content in these conditions, and of no disease is this truer than of cholelithiasis. There are those who publish almost constant hypercholesterinemic figures[11] for this disease, while others have had very irregular results.[12] [13]

In the winter of 1916-1917, hoping that a determination of the cholesterol content in the blood of gastro-enterological patients would help in diagnosis, especially when the biliary system seemed to be in question, it was planned to examine a series of cases in this way. The Bloor[14] method was employed. Whole blood was used and it was obtained during the morning hours. After a prolonged interruption in the work the number of patients so investigated reached 60 and the number of blood cholesterol readings 75 in these cases.

In this group two cases of diabetes showed 160 and 151 mg. from 100 c.c of blood, while a third case yielded values of 241 and 304 mg. The latter case has a gall-stone history and corresponds in type, but the presence of calculi has never been established. These cases exhibited no acidosis.

Two cases of pernicious anemia showed 139 and 105 mg. Both of these cases presented an achylia gastrica, and it must be assumed that the upper abdominal symptoms here in all probability were gastric in origin. The well-known antihemolytic powers of cholesterol are to be considered in connection with the anemias, and these low figures are interesting in this light. Surroeo[15] contends that cholesterol findings in the blood are a measure of the state of the organic defenses, and he is not alone in this opinion. Findings below 129 mg. show a weakness in this respect, according to this Frenchman, and findings above 200 mg. indicate a slow infection of some type. Findings below 100 mg. are of bad prognostic character.

Assuming that the leukocyte count is a measure of organic defense or an index to the presence or absence of infection, we found that the low leukocyte count cases in our series averaged 202 mg. of cholesterol, while the increased leukocyte cases yielded an average of 220 mg. A case with only a transient gastrointestinal upset gave findings of 363 mg. and 336 mg. of cholesterol and had but 5,000 leukocytes. Our lowest cholesterol finding was in a case with 100 mg., and white cell counts of 9,100 and 10,000. None of our low cholesterol cases appeared to be in grave danger nor to be lacking in defense reactions.

Carcinoma and cholesterol, according to some, present an interesting relationship. Luden[16] reports a high cholesterol content in carcinoma and that cholesterol promotes cell multiplication, especially cells of the malignant variety. Our two cases of carcinoma showed readings of 120 and 180 mg.

Four jaundice cases were of the catarrhal variety and yielded 144, 143, 214 and 171 mg., while a single obstruction case showed 330 mg. This accords with the findings of Rothschild and Felsen,[17] who report high findings in the obstructive jaundices.

In gastroduodenal ulcer our findings were 166, 169, 176, 115 and 250 mg. per 100 c.c. of blood. In three of these cases the diagnosis was made clinically, while in one the lesion was found at operation and the other showed a filling defect on Roentgen ray examination.

Intestinal stasis cases apparently uncomplicated yielded the following findings: 279, 172, 136, 125, 108, 180, 480 and 125 mg.

per 125 mg. per 100 c.c. of blood. In one case three observations were made of 185, 196 and 240 mg.

In this group of 60 cases, 36 were constipated and addicted to the cathartic habit. Their average cholesterol reading was 226 mg. The highest figure was 440 mg. and the lowest one 100 mg. Those with regular daily movements of the bowels were 16 in number and averaged 183 mg. Three cases with loose, somewhat frequent movements averaged 295 mg.

In cases diagnosed as gastric neuroses of the hypersecretory variety, findings of 160, 185, 200 and 154 mg. were found. One of these cases with 440 mg. was negative in a gastro-intestinal calculus Roentgen ray examination.

A study of the gastric contents of these 60 cases showed that 28 had a high acid curve. Their average cholesterol reading was 218 mg. The normal acid cases numbered 12, with an average reading of 220 mg. Achylia gastrica cases averaged 178 mg., while those with low acid curves showed 184 mg. per 100 c.c. of blood.

In five chronic appendicitis cases we found 180, 190, 223, 285 and 318 mg. of cholesterol in 100 c.c. of blood. The last two were operative patients in whom the biliary apparatus was declared negative, but with the appendicular condition there existed widespread adhesions throughout the right abdomen.

The following isolated diagnoses were made in cases with their respective cholesterol readings:

Gall-bladder disease, sine calculus (operative)	149 195
Rheumatoid arthritis	187
Chronic pancreatitis	248
No abdominal lesion (operative)	286 147
Visceroptoses (2)	235 216
Migraine and hypertension	210
Abdominal adhesions (operative)	100

The relationship of cholesterinemia and gall stones in our cases is shown in this table:

Gall stones (clinical).	Gall stones (operative).
206	250
151 333	190
250 280	200
316	

Gall stones (Roentgen ray).	Passed stones per bowel.
136	210
200 220	122
260	After passage of stones, calculus Roentgen ray negative.
222	
284 160	
177 260	
310 232 250	
260	
270 182	

Bracketed figures represent findings in the same case.

The question of the relationship of cholesterol and gall-stone formation has been much discussed in the literature. For the most part the evidence points to an inconstant and uncertain status. Reiman and Magoun[18] in a study of 60 operative cases found that other upper right abdominal lesions yielded cholesterol figures almost as high as those obtained in stone cases, both averages being above normal. Gorham and Meyers[19] and Denis[20] are in accord that a cholesterol estimation has little clinical and prognostic value in calculus cases.

Obviously there is no reason to assume a hypercholesterinemia in stones of other than cholesterol composition. It must also be true that the precipitation of cholesterol cannot be a uniformly progressive procedure if it depends largely upon an exogenous origin in the blood or bile; that even though a stone were one of hypercholesterinemic origin, findings in the blood would not always show high readings for this lipoid. When one subtracts the instances when stone formation has an infection or stasis origin we add still further to the instances when biliary calculi might be present without a hypercholesterinemia. If we consider the weight of gall stones and add the amount of cholesterol in the bile, together with the length of time required for their formation, it seems only reasonable to assume abnormal conditions of crystallization and precipitation as etiologic factors. The causes of crystallization seem to be found in nuclei of bacteria or epithelial cells.

Whether a hypercholesterinemia can be caused by a lesion of the biliary system, in turn due to the presence of stones, is uncertain. That lesion could scarcely be anything else than an inflammatory one, and it is well known that stones may exist without inflammation, and inflammation along the biliary tree can be very extensive and severe without calculus formation. The relationship of gall stones and cholesterol in the blood seems to be a complicated one—one which is intimately related to a complex of food, bile, blood and body tissues—their amount, chemistry, bacteriology and physical properties.

SUMMARY.

1. Sixty cases presenting gastro-enterologic aspects were studied for their blood-cholesterol content.
2. In so far as possible the clinical, Roentgen ray and postoperative findings are correlated.
3. The relationship of constipation, hyperacidity and leukocytosis to cholesterol findings are considered.
4. The highest finding was 480 mg., the lowest was 100 mg.
5. Inconstant results were obtained in most of the conditions when more than one case was studied.
6. The figures for cholelithiasis averaged high (228 mg.). Other cases averaged 207 mg., both averages being above the high normal limit of 200 mg. The highest finding was 316 mg. and the lowest 136 mg. The highest reading in noncalculus cases was 480 mg. and the lowest 100 mg.
7. Inconstant findings were sometimes found when more than one estimation was made in the same case on different days.

CONCLUSIONS.

From our limited studies and an estimate of the literature, blood cholesterol seems to offer little practical diagnostic help in gastro-enterological cases.

Appreciation is expressed to Dr. Joseph Sailer for the use of clinical material and to Miss Sarah C. Hetherington for her valuable assistance.

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SPONTANEOUS AND OPERATIVE CURE OF CIRRHOSIS OF THE LIVER.

REPORT OF ILLUSTRATIVE CASES.*

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Some writers give such a gloomy prognosis of cirrhosis of the liver that I am prompted to report a few cases, the outcome of which justifies a greater optimism. One of these cases is so remarkable that I venture to describe it at some length.

Report of Case With Spontaneous Cure.

Case 1.—David G., a boilermaker, aged 57, had been a very hard drinker all his life. For years his daily consumption was a quart of beer and whiskey mixed. He began to use tobacco at the age of seven, and always used it to excess. He was admitted to the surgical wards of the Philadelphia General Hospital, October 11, 1910, on account of a fracture of the left wrist, and was later transferred to the medical wards with a diagnosis of cirrhosis of the liver. When I saw him he had marked ascites with general enlargement of the veins of the abdomen and a very typical caput medusae. In addition to the fracture of the wrist there was also an osteo-arthritis of the right hip which rendered him very lame. Soon after his admission to the medical wards he had a profuse hemorrhage, in all probability from the lower end of the esophagus or from the stomach. Edema of the legs set in about the beginning of November. At the same time the abdomen became more tense, but the patient refused for a long time to be tapped. Finally he yielded. After the tapping, the liver and the spleen could be plainly felt. From this time on, February 6, 1911, the patient had to be tapped at varying intervals, the longest being twenty-four days, the shortest nine days. At each tapping, except the last one, from three and one-half to four and one-half gallons of straw-colored fluid were removed. We left the time for the tapping to the patient's own judgment; he would always wait until his distress became unbearable. Despite the tapping the edema of the legs steadily increased until the limbs became of such enormous size that they could not be lifted or moved in the slightest degree. The scrotum was also greatly swollen. Toward the end of November, the patient grew irritable and peevish, and objected to every disturbance. At the beginning of December a persistent hiccup set in; about this time the irritable mental state gave place to a stupor. The patient lay constantly with his eyes shut; his face was sallow and of an ashen pallor.

We did not think that he could live more than a few days. December 11th, he was tapped for the thirty-sixth time, a gallon and a half of fluid being removed.

One day while I was making a casual examination—we had all kept aloof from him on account of his ugly disposition—I detected over the abdomen, particularly in the region of the liver and spleen, an exquisite friction, appreciable both to touch and to the ear. Over this area of friction there was also considerable tenderness. I then learned that the man had had much pain subsequent to the last tapping, and had been getting a small daily dose of morphine for its control.

To our utter astonishment the abdomen did not fill up again. Not only was there no return of ascites, but all the edema of the hugely swollen legs and of the scrotum disappeared, and the man soon began to show again his pristine amiability. Eventually he was able to be up and about, and even left the hospital, to which, however, he returned, being unable, on account of his lame hip, to earn a living.

I have seen him many times since and have exhibited him in my lectures. There is no ascites; the venous distention and the caput medusae have disappeared; the liver and the spleen are of normal size, and but for the osteoarthritis the man is perfectly well. As far as I know he has not touched alcohol since his first admission to the medical wards at the hospital.

The urine during his sojourn never exceeded 40 ounces a day, despite the use of many diuretics, but after the last tapping it increased wonderfully, reaching as high as 105 ounces a day without any drugs. The specific gravity varied from 1.010 to 1.020; occasionally there was a trace of albumin; usually the urine was chemically and microscopically normal. The blood, about the middle period of his illness, showed: hemoglobin, 65 per cent.; red cells, 3,910,000; white cells, 6,800. The differential blood count revealed polymorphonuclears, 83 per cent.; small lymphocytes, 11 per cent.; large lymphocytes, 6 per cent.

The total amount of fluid removed in the 36 tappings we estimated as from 140 to 150 gallons, or, in round numbers, nearly 1,200 pints, which, considering the specific gravity of the fluid, amounts to more than 1,200 pounds. This amount, representing about ten times the man's own weight, was removed in the space of nine months.*

The man's history and symptoms were typical of cirrhosis of the liver. He had no history of syphilis; in fact, no history of anything except extreme alcoholism. The edema of the legs and of the scrotum did not impress us being due to cardiac disease. We were at first inclined to attribute it to thrombosis of the lower vena cava, but its spontaneous disappearance negatived that view. Probably pressure on the vena cava, aided by a general weakness of the circulation, was responsible for it.

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*In A. J. C. Hare's "Walks in London," 1: 311, I came across this legend, taken from the tomb of Dame Mary Page (1728): "In 67 months she was tapped 66 times and had taken away 240 gallons of water without ever repining at her case or ever fearing the operation." This is a better record than that of David G.

How are we to explain the almost miraculous cure? Evidently, as indicated by the extensive friction, pain and tenderness, a fibrinous peritonitis followed the last tapping.*

The adhesions resulting from this peritonitis constituted a spontaneous Talma operation and sufficed to establish an adequate collateral circulation.

Report of Cases With Cure by Operation.

The other cases illustrate cure by the Talma operation.

Case 2.—S. G., aged 53, married, a retired iron merchant, native of Indiana, residing in Buffalo, came under my observation September 14, 1915. His father died of diabetes at the age of 75; his sister has diabetes and gall stone disease. His wife has never been pregnant. The patient has smoked to excess, but has never used alcohol or tea, and coffee only in moderation. He has never had syphilis, and the Wassermann test, which has been taken a number of times, has always been negative. During the preceding twenty years he had had attacks that from his description I should call biliary colic. In 1904, while in Edinburgh, he had a violent chill followed by jaundice, itching of the skin, and clay colored stools. Dr. Lauder Brinton diagnosed malaria. Ten years ago traces of sugar were found in the urine. Four years later he received some hypodermic injections, perhaps of mercury, which made him feel better.

In June, 1915, after eating three bananas, he had a violent attack of colic. Soon afterward the abdomen began to fill with fluid and it was necessary to tap him. He was tapped four times, at intervals of from ten days to two weeks; at each paracentesis from four to seven quarts of fluid were drawn off. At the end of August he was sent by his physicians to the Mayo Clinic, where a laparotomy was performed; the liver being found the seat of a manifest cirrhosis, a Talma operation was done. The patient was brought to me about a month later. I found the liver enlarged and finely nodular. The spleen was also enlarged and the abdomen was full of fluid. There were no hemorrhoids or caput medusae. October 7th he was tapped, and more than six quarts of straw-colored fluid were removed. After that the liver could be felt a hand's breadth below the costal arch; it appeared to be tough and leathery. In a little while fluid could again be demonstrated, but it did not reach any large amount. Eventually it became absorbed so that no further tapping was required. A year after the operation the patient was able to walk five miles without the slightest trouble and now, nearly four and a half years since the operation, he is well except that the urine occasionally contains traces of sugar and of albumin, with stray hyaline casts. The spleen is no longer palpable, and the liver has receded to the costal margin. Only one attack of abdominal pain of the nature of biliary colic has occurred since the operation.

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*It is not without interest that the interne who did the tapping was at that time on service in the erysipelas wards of the hospital.

CASE 3.—C. H. M., aged 62, born in Philadelphia, a house painter until a year and a half before I saw him, and since then a night watchman in a bakery, had been a heavy drinker of beer and whiskey for 20 years; he had also consumed large amounts of tobacco. His wife was in the insane department of the Philadelphia General Hospital. He had never had any venereal or other serious disease, except an attack of influenza in the fall of 1918 which lasted about four weeks, but did not confine him to bed. He never had painters' colic or any symptoms of lead poisoning. He had always enjoyed excellent health.

In February, 1919, without any apparent cause, the abdomen began to swell. Getting no relief from treatment, he came to the Philadelphia General Hospital, April 20th. He had the general appearance of a patient with cirrhosis of the liver. The muscles were rather wasted. The weight on admission was 140 pounds; his best weight had been 170 pounds. The heart presented nothing abnormal. Over the bases of the lungs posteriorly, a few râles could be heard. The abdomen was greatly distended, the veins much enlarged. The liver and spleen could not be palpated. May 3rd he was tapped, four and one-half gallons of straw-colored fluid being removed. After the tapping, the liver could be felt three inches below the costal margin; it was hard, finely nodular and tender. The spleen was not palpable. From that time on until October 22nd, he was tapped seventeen times at intervals ranging from four to 23 days, a total of nearly 50 gallons of fluid being withdrawn.

The urine was examined on 12 occasions and showed usually a few hyaline and granular casts, a trace of albumin and occasional leukocytes. The specific gravity varied from 1.011 to 1.025. The phenolsulphonephthalein test was made four times with rather varying results; the elimination in the first hour was from 8 to 30 per cent., in the second from 5 to 20 per cent., and the total from 13 to 50 per cent. The Wassermann test with blood, spinal and peritoneal fluid was always negative. A blood count revealed: red cells, 4,800,000; white cells, 13,000; hemoglobin, 90 per cent. The blood sugar, after the administration of 100 gm. of dextrose at 11 a. m. (breakfast at 6 a. m.) was: at 11 a. m., 0.08 per cent.; at 12 m., 0.15 per cent.; at 1 p. m., 0.14 per cent. The peritoneal fluid was sterile and contained only a few leukocytes and epithelial cells.

As the patient was losing ground and had begun to dread the tapping, it was easy to persuade him to submit to an operation. This was performed under ether anesthesia, October 22nd, by Dr. T. Turner Thomas, of the surgical staff of the hospital. Just preceding operation, the patient was tapped, and about nine quarts of fluid removed from the abdominal cavity. A right rectus incision about four inches long was made over the gall-bladder. The remaining fluid was removed from the abdomen with towels. The upper surface of the liver and spleen and adjacent peritoneum was rubbed with gauze until blood exuded slightly. The omentum was sutured to the anterior abdominal wall with four catgut sutures, and the lower portion of the omentum was sutured in the abdominal incision just beneath the skin. The incision was closed in the usual way. A short median line incision was made just above the pubis, and a rubber drainage tube inserted into the pelvis. On direct inspection during the operation, the liver was found somewhat enlarged, deeply congested and finely granular.

Convalescence was uneventful except for considerable gaseous distention of the abdomen and some looseness of the bowels. November 12th the sutures were removed from the upper incision; healing was complete and clean. December 23rd the patient was retransferred to the medical wards, from which after a short stay he was dismissed virtually well.

COMMENT.

The first case illustrates the wonderful *vis medicatrix naturae.* The other two cases show what may be accomplished by surgical means.

Cirrhosis of the liver is one of the few chronic nonbacterial viscereal diseases—perhaps the only one—that may actually be cured. It is interesting that the cure of one of its outstanding symptoms, namely, the ascites, brings about an arrest or cure of the cirrhotic process. We have been accustomed to look on hepatic cirrhosis as a progressive disease, and on ascites as a symptom of an advanced and, to all intents and purposes, incurable stage. Yet, as the cases here reported and many others prove, after the ascites is abolished, not only does the cirrhosis make no further progress but the damage that has been done to the liver seems to be compensated to a sufficient degree to permit a virtual restoration to health. When the cirrhosis is dependent on syphilis, such a striking therapeutic result is more easily comprehended. But in none of the cases here recorded could syphilitic infection be demonstrated.*

The cure of cirrhosis by the Talma operation cannot be due merely to the establishment of an adequate collateral circulation. That would be too mechanical a view of the matter. The liver itself must undergo a change, and probably also the spleen, the share of which in the pathogenesis of cirrhosis is, I believe, much greater than is generally conceded.

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*Polyserositis, which exhibits a tendency to spontaneous cure, closely simulates cirrhosis of the liver. The three cases here reported gave no sign of being anything more than cirrhosis, evidence of general serous membrane involvement being lacking.

I have seen the Talma operation fail, owing, I think, to my having postponed it too long.

If the intervals between tappings are decreasing, there is no use in waiting. The chances for a fortunate outcome from tapping alone, as in the first case, are so slight, and the action of drugs is so uncertain, that the patient should have the benefit of an operation if the circulation and the kidney function are such that the operation *per se* is justified. The operation is best performed when the abdomen is full of fluid. One should not be discouraged if the result is not immediately apparent, and if the patient has to be tapped once or twice afterward. As Case 2 shows, the result may nevertheless be entirely satisfactory.

DISCUSSION.

Dr. Charles G. Stockton, Buffalo: I see that my name is down on the program to open the discussion on this paper, so I shall take the liberty of speaking first. The cases reported by Dr. Riesman are instructive and extremely interesting. It should be noted that he speaks not only of the disappearance of the important symptom of ascites, but also of the clinical cure of his cases. Now I have seen a good deal of the Talma operation and its results, through the enthusiastic co-operation of my colleague, the late Roswell Park, who worked with me on that subject for some years. We had several successes that seemed to me flattering, and more failures. Undoubtedly it is true that the successes did not depend entirely on the reestablishment of large inosculations, as has been suggested by Dr. Riesman; for I remember a case in which there was the most extraordinary development of superficial veins that I have ever seen, except in obstruction of the inferior vena cava, in which we sometimes have a very picturesque abdomen resulting. This patient had a most extraordinary inosculation, but his ascites was unchanged. He remained in bed for two years, and was tapped about once in three weeks. I have records of the amounts taken from this man. I cannot recall them now, but they were in excess of those mentioned by the speaker. He was never, so far as I know, cured of ascites, but he was sometimes benefited by a somewhat intensive antisyphilitic treatment. This was given, not because he had a Wassermann, for it was negative; but because I became convinced from study that, when you see cirrhosis of the liver in hospital practice, you almost always suspect that syphilis of the liver plays a part in the condition. The cirrhosis that I find in ward cases is more often syphilitic than alcoholic; and this is not revealed by the Wassermann as often as are other syphilitic lesions. The question is whether the removal of the ascites and the inconvenient abdominal pressure, and the nutritive disturbances entailed thereby, means a cure of the disease. Is a case of cirrhosis of the liver cured, because of the disappearance of its prominent symptoms? To my mind, this is improbable. There may be restoration of tissue, as Dr. Riesman suggests (and in regard to that matter, I bow to his larger experience); but in my own experience I have never seen patients with cirrhosis of the liver recover good general health. I have known them, however, to go on with improved health, and for many years. An old medical friend of mine, who recently died at the age of 80, came to me with a diagnosis of cirrhosis of the liver made by the late A. L. Loomis. His liver could not be made out by percussion. The spleen was enlarged, but there was no ascites. He suffered from the intoxication that we are in the habit of attributing to cirrhosis, but a careful diet led to the disappearance of all evidences of cirrhosis except the physical signs and the occasional recurrence of intoxication; yet he lived to an old age. On the other hand, I have seen many cases improved by simple antisyphilitic treatment. These patients got along in fairly good health and were able to return to work; but I never considered them well. It may have been syphilitic cachexia that remained after treatment. I have seen cure follow repeated tapping. Austin Flint called attention to the fact that repeated aspiration does seem to produce cure of cirrhosis of the liver, and reported cases similar to those reported today by Dr. Riesman.

The question of the inosculation established by the Talma operation is most important. Just how much good it does is hard to decide. I am convinced that it occasionally does good; but I think that by this treatment we introduce a new and uncertain element, that is the presence of portal blood in the systemic circulation. I read a paper on this question before the American Medical Association in 1880, at the meeting at Newport. The views there expressed I still consider to be sound. I should like to have Dr. Riesman tell us what benefit he thinks accrues from the Talma operation. If it does not come from reestablishment of the circulation, what does he think influences toward recovery?

I cannot get it into my mind how the function of the liver can be changed. A change so marked as that which we find by pathological study of the livers of cirrhosis cases seems to me to preclude any actual recovery from the disease.

DR. JULIUS FRIEDENWALD, Baltimore: Dr. Riesman's paper is extremely interesting. There are two cases that I should like to report to the Society in this connection, one of spontaneous cure and the other of operative cure in cirrhosis of the liver.

The first case is that of a man 45 years of age, a habitue of alcohol, who had been troubled for several months with indigestion, nausea and vomiting, and, finally, with abdominal swelling. On examination, he was found to have all the typical signs of cirrhosis, with an enormously distended abdomen full of fluid. After the withdrawal of the fluid, an indefinite mass was noted in the epigastrium. At operation, a typical atrophic cirrhosis of the liver was found. The patient collapsed under the anesthetic, so that no further steps were undertaken, and the abdomen was closed. The patient recovered, however, and but little of the fluid returned. He gradually gained strength, and was well for over two years, after which he could not be located.

The second case is that of a physician, who was operated on by Dr. McGlannan in July, 1914, for cirrhosis of the liver. An omentopexy was done and the patient has since been about, practicing medicine and apparently well, having had no further fluid in his abdomen. He has since had, however, occasional hemorrhages from the stomach.

Dr. Allen A. Jones, Buffalo: I have a case that it might be of interest to mention in connection with these interesting cases reported by Dr. Riesman. This man came to me last autumn with very little fluid in the abdomen. I watched him until he collected enough and then he was tapped. We tapped him six times, and then at one tapping injected oxygen into the peritoneal cavity and took an X-ray to see whether we could thereby find an abdominal tumor. The result was negative so far as tumor was concerned. Three months ago he was operated on and found to have a cirrhotic liver, the peritoneum was stripped from the abdominal wall and the omentum sewed in on each side. For four days after the operation he was quite soporous; but he came along all right. It has not been necessary to tap him since. There is some fluid present. His legs are somewhat swollen, but on the whole he is doing very much better than before the operation.

Dr. Arthur F. Chace, New York City: It seems to me that the prognosis for spontaneous cure in cirrhosis of the liver, as far as the ascites is concerned, depends on whether the ascites is due to obstruction in the portal circulation or to a proliferative peritonitis. Those cases of ascites in cirrhosis of the liver in which the fluid is caused by obstruction of the portal circulation, in my opinion, never get well; the patients die after a few tappings. In those cases of spontaneous recovery, the fluid is due to proliferative peritonitis. They recover by developing a fibrous peritonitis. This is one reason why the Talma operation occasionally succeeds, not in relieving the obstruction to the portal circulation but in stimulating fibrous peritonitis. In the case that Dr. Riesman reported first, it seems to me, there was a factor of renal and cardiac insufficiency to be considered. We must consider how much fluid was due to cardiac insufficiency and how much to renal. I am inclined to think that only a little was due to portal obstruction.

Dr. John A. Lichty, Pittsburgh, Pa.: Two points have come out in this discussion that rather surprised me. First, when Dr. Riesman spoke so surely of a recovery in these cases in so short a time. May there not be a return of the ascites later?

Dr. Riesman: The time in one case was four years.

Dr. Lichty: I thought that it was only a year ago that one was operated. Second, the relation of anti-luetic treatment to these cases. Nearly all cases of cirrhosis of the liver have at some time or other a turn at antiluetic treatment; yet so few of them get well, that I should not like to desert the idea that alcohol is more of a factor than the luetic infection.

Finally, I should like to speak of a method of treatment that we have adopted at the Mercy Hospital in Pittsburgh. It gives better results, when it works, than any method that we have yet undertaken. That method consists in implanting the saphenous vein into the peritoneal surface—dissecting it out from the thigh, slipping it up under the skin, and implanting it in the peritoneal surface. We have had to my knowledge five patients operated at the Mercy Hospital in the last five years. Two did not do very well, but all the rest did well. They have left the hospital and are going on with their usual work, and they seem to be well. The last was two years ago; but how permanent the improvement may be I do not know. In one of these, I will say for the benefit of Dr. Stockton, we used antiluetic treatment very definitely because we had a positive cholesterin reaction; and on that ground we gave mercury and the iodides and also arsphenamine. Two are at work now and seem well.

Dr. White: If there is no further discussion of this paper, I will ask Dr. Riesman whether he has anything to add.

Dr. Riesman (closing): With regard to the question of Dr. Lichty as to the length of the time of the spontaneous cure, I would say that it has now lasted nine years. Of the operation cases one was operated on four and a half years ago; the other last October. In this latter case, the man was very sick when he came in to the hospital and went out, as far as we could tell, perfectly well. I have had no experience with the implantation of the saphenous vein, but have heard that it gives good results.

Most hospital patients having cirrhosis receive iodid of potassium at some time; very few respond. The syphilitic liver is a large liver; and in the earlier stages gives the impression rather of malignant disease than of cirrhosis. I have at present such a case under observation—a diagnosis of carcinoma of the liver had been made. The organ extended down to the umbilicus and was evidently the seat of large gummatous masses. Under anti-syphilitic treatment the liver returned to normal conditions. The patient never had ascites.

The question that Dr. Stockton has asked as to how I would explain the cure after the Talma operation has been partly answered by Dr. Chase. In some cases, no doubt, there is a serositis, not necessarily a polyserositis. The man in whom the cure was spontaneous presented the typical picture of alcoholic cirrhosis. He had no cardiac lesion. He has now been perfectly well for nine years. There is nothing to indicate that he has ever been sick. My thought is that upon the establishment of an adequate collateral circulation, some of the toxic substances that are the cause of irritation of the liver no longer pass through the organ. The liver no longer receiving this material, has a chance to get functional rest and in that way regains sufficient power to perform its complex processes. It must be acting in these patients or they would not be as well as they are.

Dr. Friedenwald's two cases are interesting and very relevant to this discussion.

Polyserositis has been alluded to by Dr. Chase. In a general way, when many tappings are required, the chances are that the case is one of chronic peritonitis. Dr. Chase says that true cirrhosis cases are seldom tapped more than a few times. I am not able to agree with him in this statement.

I believe I have answered most of the questions raised. The important point is not to delay the operation too long, but to resort to it as soon as it is evident that neither specific treatment nor tapping will cure the patient.

OBSERVATIONS ON THE BEHAVIOR OF THE NORMAL PYLORIC SPHINCTER IN MAN.*

By C. W. McClure, M. D.,

and

L. Reynolds, M. D.

Boston.

Experimental studies on the physiological factors influencing the motor functions of the gastrointestinal tract and the relation of these motor phenomena to the digestion and absorption of food products have been largely carried out in the lower animals. But the results obtained from such studies can be applied only in a general way to the physiology of the digestive tract in man. It follows that more intimate knowledge concerning various phases of the physiology of the alimentary canal in man would aid in the interpretation of results obtained from experimental studies of the pathological physiology of human digestion. For this reason certain phases of the motor phenomena of the normal stomach in man are being studied. The present preliminary report deals largely with work bearing on the question of "acid control" of the pyloric sphincter in man.

Cannon[1] elaborated the theory, suggested by Pawlow[2] among others, that acid controlled the action of the pyloric sphincter in animals. In epitome this theory is that the presence of acid in the antrum of the stomach causes relaxation of the pyloric sphincter, while the presence of acid in the duodenum causes the sphincter to contract. The evidences on which this theory is based were obtained from experiments carried out on the lower animals. There are, however, phenomena occurring in the lower animals which the theory of "acid control" does not explain, and for this reason other controlling factors are assumed to exist. Whether or not acid or some other factor exerts the principal control of the pyloric sphincter in man has not been demonstrated, and with this problem the present investigation is concerned. The results obtained by previous workers will be discussed later.

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*From the Medical Clinic and Radiographic Department of the Peter Bent Brigham Hospital, Boston.

[1] Cannon, W. B.: The Acid Control of the Pylorus. *Amer. Jour. Physiol.*, Boston, 1907, XX, 284.

[2] Pawlow: The Work of the Digestive Glands. London, 1902, 165.

EXPERIMENTAL PROCEDURES.

1. The degree of contraction of the pyloric sphincter when foodstuffs partially fill the normal stomach was studied.

The data for this were obtained from fluoroscopic observations.

2. The behavior of the sphincter relative to the passage of protein or of fatty foods from the normal stomach into the duodenum was studied.

This was determined by fluoroscopic observations of the stomachs of young men after feeding meals composed of ground lean meat or of fatty bacon and egg yolks. The two kinds of meals were of constant bulk and consistency. In order to be opaque to the Roentgen rays, they were intimately mixed with barium sulphate.

3. Effect on the pyloric sphincter of direct application of HCl and $NaHCO_3$ solutions to both its antral and duodenal ends, as observed by means of the fluoroscope, was studied.

For this purpose a Rehfuss gastro-doudenal tube was used. It was found that the placing of the metal bucket in the desired area was largely a matter of luck. In the course of several attempts a few successful experiments were accomplished. In these experiments N/40 HCl, N/20 HCl, N/10 HCl and 1 and 5 per cent. $NaHCO_3$ solutions were introduced into different portions of the duodenum. The solutions were warmed to body temperature before their introduction.

1. *The Study of the Degree of Contraction of the Normal Pyloric Sphincter.*—That the pyloric sphincter is closed when food is in the stomach, except during the periods in which it opens to permit the ejection of food into the duodenum, was demonstrated in 1913 by Cole.[3] His observations were so readily and easily confirmed by fluoroscopic studies that they are now universally accepted. In this way the authors have confirmed Cole's observations a large number of times. Further evidence that the normal quiescent sphincter is in a state of contraction is afforded by the fact that barium mixtures can be forced from the

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[3] Cole, L. G.: Physiology of the Pylorus, Pilleus Ventriculi and Duodenum as Observed Roentgenographically. *Jour. Amer. Med. Assoc.*, Chicago, 1913, LXI, 762.

stomach into the duodenum only by the use of considerable pressure on the abdominal wall, and can be accomplished probably only when the sphincter has opened in relation to the advance of an antral peristaltic wave. When it can be accomplished the filled antrum and first portion of the duodenum are seen to be connected by a narrow isthmus of barium, which represents the sphincter; on cessation of pressure this narrow isthmus disappears.

2. *The Study of the Behavior of the Pyloric Sphincter Relative to the Passage of Carbohydrate, Protein and Fatty Foods into the Duodenum.*—In the routine, clinical X-ray examination of the stomach a carbohydrate meal consists of 500 c.c. of malted milk in which 90 gms. of barium sulphate are held in suspension by the aid of potato starch. The meal starts through the normal pyloric sphincter from at once to within a few minutes after it has been ingested. The normal sphincter opens regularly as each antral peristaltic wave approaches, and thus permits the passage of the barium meal into the duodenum. In two normal young men the same phenomena occurred after the ingestion of thick oat-meal porridge mixed with barium. Except in abnormal conditions accompanied by pylorospasm, opening of the pyloric sphincter at irregular intervals, as described by Cannon in cats, was not observed. The regular opening and closing of the pyloric sphincter, except in the presence of pylorospasm, is so well known (3) that no further work on carbohydrate meals was attempted in the present investigation.

Obervations on the behavior of the pyloric sphincter after the ingestion of protein food were made in young men, eight normal, one with a quiescent duodenal ulcer, and one woman whose physical condition was normal. Similar observations were made after the ingestion of fatty food in five of the normal young men. The results obtained are illustrated by the following protocol:

PROTOCOL I.

L., white, male, student, aged 25.

Meat Meal.	*Fatty Meal.*
Meat and barium meal with 100 c.c. of water to drink.	Fatty and barium meal with 100 c.c. of water to drink.
5:25 p. m. Began eating the meal.	6:07 p. m. Began eating the meal.

5:35. Finished eating the meal.

Fluoroscopic observations; subject in reclining position.

5:35-38. Gastric peristalsis active. As each wave approached the pyloric sphincter barium passed through in normal amount[*] into the duodenal cap.

5:45-47. Ditto.

5:53-55. Ditto.

6:00-05. Ditto.

6:14. Finished eating the meal.

The meal was nauseating to patient, since he did not like fat; no nausea present after having eaten the meal.

Fluoroscopic observations; subject in erect posture.

6:14-16. Gastric peristalsis active. As each wave approached the pyloric sphincter barium passed through in normal amount into the duodenal cap.

6:21-24. Ditto.

6:29. Ditto.

RESUMÉ OF THE FINDINGS OUTLINED IN PROTOCOL I.

The amounts of foodstuffs and of water fed in these experiments, illustrated in the foregoing protocol, formed a very thick mush when thoroughly mixed together in a mortar. To obviate the criticism that the consistency of the food permitted it to act more like liquids than solid food, the meat and the fatty meals were given to subjects without the addition of water. The results obtained were similar to those reported in the above protocol. (Protocol I.)

Observations similar to the one illustrated in the foregoing protocol demonstrated that either fatty or protein meals began to leave the stomach within from 3 to 10 minutes after the food was first swallowed. Furthermore, the food passed through the pyloric sphincter into the duodenum as each and every peristaltic wave approached that orifice. In the same subject no difference in the amounts, either of the same food or of the different foods which passed through the sphincter at different times, was observed. These same phenomena were demonstrated throughout observations made at intervals for periods of six hours on four of the normal young men after the ingestion of the protein meal and in one after the ingestion of the fatty meal.

After feeding either carbohydrate, protein or fatty food, the barium containing chyme flowed through the sphincter as an antral peristaltic wave approached that orifice and continued to do so until the wave had spent itself. Therefore the sphincter did not close immediately upon the entrance of chyme into the duodenum.

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[*] By "normal amount" is meant that quantity of the barium which passes through the pyloric sphincter after the ingestion of the barium meal commonly used in X-ray examinations for diagnostic purposes.

3. *Effect of HCl and Sodium Bicarbonate Solutions on the Pyloric Sphincter, as Observed Through the Fluoroscope.*—The effect on the opening and closure of the pyloric sphincter produced by the direct introduction into the duodenum of solutions of HCl, and in a few instances of 1 and 5 per cent. $NaHCO_3$ solution, was ascertained, the behavior of the sphincter being observed through the fluoroscope. The functioning of the sphincter was determined by the passage or nonpassage of barium from the stomach into the duodenum.

The conditions under which the experiments of this division of the work were performed are as follows: The doudenal tube was given subsequent to a 12-hour fast; the tube was allowed to pass into the duodenum. The time required for this to occur was variable. The subjects used were hospital patients whose physical condition at the time of the experiments was considered to be normal. The usual clinical gastric analyses of these subjects showed no abnormalities. After the tube had entered the duodenum a meal was fed. The meal used was a meat and barium meal either rinsed down with 200 c.c. of water or made into a thick mush by grinding in a mortar with water. In certain cases a malted milk, potato starch and barium mixture were also given.

In these experiments N/40, N/20 and in one case N/10 HCl solutions, warmed to body temperature, were injected in from 5 to 20 c.c. amounts into the first, second or third portions of the duodenum. The time of the injection in relation to the normal opening time of the pyloric sphincter varied. The injections were made (1) just prior to the normal opening time of the pyloric sphincter in relation to the advance of an antral peristaltic wave, or (2) the injection of 20 c.c. of the acid was begun at the time just mentioned and continued until after the sphincter had opened consecutively for a second or third time. Besides the acid solutions, 1 and 5 per cent. sodium bicarbonate solutions were used under the same experimental conditions. In four of the experimental subjects no effect was observed on the opening time of the sphincter, the amounts of barium passing through it, or upon its time of closure after the introduction of either the acid or alkali. In a fifth subject the introduction of N/20 HCl or of 1 per cent. $NaHCO_3$ into the third portion of the duodenum produced no effect on the opening or closure of the pyloric sphincter; but the introduction of N/20 HCl into the first portion of the duodenum produced either complete or partial pylorospasm, and vigorous duodenal antiperistalsis. In a sixth subject the introduction of N/20 HCl into the third portion of the duodenum produced either complete or partial pylorospasm, associated with marked diminution in the depth of gastric peristaltic waves; while the introduction of 1 per cent. $NaHCO_3$ solution produced no observable effect of any kind. In two subjects movements of the duodenal tube, such as pulling the metal tip from the third portion into the first part of the duodenum, produced pylorospasm, associated with the development of very shallow gastric peristaltic waves.

In one subject a loop of the duodenal tube passed through the sphincter into the duodenum in such a way that the metal tip was held close against the antral end of the pyloric sphincter. This permitted solutions to be poured directly onto the antral end of the sphincter. The introduction of N/20 HCl on two occasions produced pylorospasm, associated with very shallow gastric peristaltic waves. Under the same experimental conditions the introduction of 1 per cent. $NaHCO_3$ solution produced no effect on the pyloric sphincter or on gastric peristalsis. That some of the acid did not enter the duodenum is made probable by the facts that (1) the sphincter was in a state of contraction at the time of introducing the acid, and (2) the barium residue surmounting the duodenal end of the sphincter was not disturbed by introducing the acid.

The production of pylorospasm, of very shallow gastric peristaltic waves and of duodenal antiperistalsis was interpreted as the result of abnormal irritation either of the duodenal or of the antral mucosa, depending upon the position of the metal tip of the tube, or of the pyloric sphincter.

Discussion.

Observations such as the one outlined in Protocol I, made after feeding the different foodstuffs, demonstrate that, regardless of the type of foodstuff ingested, finely divided foods begin to leave the stomach within a period varying from a few to about 10 minutes; that is, barium was passing through the sphincter by the time the subject could ingest the meal and be prepared for fluoroscopic observation. That this phenomenon is true for carbohydrate meals has already been stated. But with the exception of Cole,[4] no observations in man of other types of meals have been published, and his publication unfortunately gives no experimental details. The barium meal passed through the pyloric sphincter as each antral peristaltic wave approached that orifice. Cole had previously made similar observations. The opening of the pylorus at irregular intervals, as described by Cannon in cats, was not observed. Since during this interim but one peristaltic wave swept along the antrum, the contents of that region were not undergoing a mixing process, and for this reason the same food must have remained in contact with the gastric mucosa. Therefore the surface of the antral mucous membrane would be covered with material of a fairly constant degree of acidity. It is very probable that the reaction of this material is acid. This statement is based upon the following facts:

1. The authors have found in two subjects, after feeding a meat meal, the food when first ejected from the stomach into the duodenum to contain free HCl, in the strength of N/60 and N/500. The position of the metal tip of the duodenal tube and the phenomena transpiring in the stomach and duodenum at the time the specimen for analysis was withdrawn were determined by fluoroscopic observation.

2. The contents aspirated from the stomach after feeding the Riegel[5] test meal of meat and potatoes are acid, and also after feeding various kinds of meats.[5]

3. HCl is constantly present in the stomach after feeding the Ewald test breakfast of bread and water.

From the above it follows that in order for the stimulation of acid in the stomach to open, and in the duodenum to close, the sphincter there must be assumed the existence of a very delicate balance between the antral and duodenal reflexes in relation to the presence of the same degrees of acidity, the duodenal reflex being much the more sensitive.

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[4] Cole, L. G.: *Amer. Jour. Physiol.*, Boston, 1917, XLIII, 618.

[5] Riegel, F.: *Die Erkrankungen des Magens.* Wien, 1903, I, 123. Fishback, H. R., Smith, C. A., Bergheim, O., Lichtenthaeler, R. A., Rehfuss, M. E., and Hawk, P. B.: Gastric Response to Foods. *Amer. Jour. Physiol.*, 1919, XLIX, 174, 204, 222 and 254.

McClendon and Meyers[6] and the authors have found the contents of the first portion of the human duodenum to be either neutral or of low acidity. After emptying itself of food just ejected from the stomach the duodenum will, therefore, be practically neutral in reaction, and in the presence of pancreatic juice even alkaline. Then, if acid in the stomach causes the sphincter to relax, the sphincter should become patent when the duodenum empties itself of food. That this is not the case is demonstrated by the difficulty experienced in pushing barium through the sphincter into the duodenum by palpation of the abdominal wall. In fact, it is probable that barium can be caused to enter the duodenum by this means only when the sphincter opens in relation to the advance of an antral peristaltic wave. From this it follows that, if the pyloric sphincter is under "acid control," then the fact that it opens at regular intervals and normally only in a definite relation to the advance of an antral peristaltic wave makes it necessary to assume: (1) either that there is a finely adjusted acid regulatory mechanism in the antral region of the sphincter, which produces the proper degree of acidity to relax the sphincter at a fairly exact time in relation to the approach of a peristaltic wave toward the sphincter; or (2) that a comparable mechanism for properly neutralizing the contents of the duodenum must exist. If either or both such mechanisms exist, they must be so adjusted as to permit of changes in the rhythm of the time of opening of the sphincter as occurred in one of our experiments after pouring alkali onto the antral end of the pyloric sphincter. Furthermore, while the sphincter closes suddenly, it remains open an appreciable length of time. Barium flows through the sphincter as an antral peristaltic wave approaches and continues to do so until the wave has spent itself. The sphincter, therefore, does not close as soon as acid enters the duodenum, and the presence of acid we have demonstrated experimentally in two subjects. If acid in the duodenum causes closure of the sphincter, then (1) either the proper degree of acidity must be developed always at a time when an antral wave has spent itself, or (2) a secondary mechanism in some way regulating the reflex must be assumed.

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[6] Myers, F. J., and McClendon, J. F.: Note on the Hydrogen Ion Concentration of the Human Duodenum. *Jour. Biol. Chem.*, New York, 1920, XLI, 187.

The existence of the various factors outlined are necessary to explain the theory of "acid control" of the sphincter in man and their existence has not been proved. The assumption of the existence of these factors renders the mechanism of the control of the sphincter exceedingly complex. It is possible that some other less complicated mechanism may exist in man. The latter has been suggested, but not proved, by Luckhart, Phillips and Carlson.[7] These investigators found a relation existed between the muscular activity of the antrum and the opening of the sphincter.

In four of the experiments here reported it has been shown that acid introduced into the duodenum did not prevent the opening of the sphincter. These observations do not support the theory that acid in the duodenum causes contraction of the pyloric sphincter. Furthermore, sodium bicarbonate was poured into the duodenum in a quantity which, judging from the limited observations of the degrees of acidity of duodenal contents, was sufficient to bring about neutralization. The latter must have occurred in an experiment in which 5 per cent. $NaHCO_3$ solution was poured into the duodenum. The carbonate solution was introduced on two occasions in quantities of 10 c.c. and 20 c.c. after the pyloric sphincter had opened and up to the time of its complete closure. Before the introduction of the carbonate solution the chyme was collected as it was poured into the first portion of the duodenum during the latter half of the time the sphincter remained open. This allowed the first portion of the chyme to clear the region of the metal tip of duodenal contents. The concentration of free HCl in the chyme was N/500, and of total acidity N/25 (titrated to phenolphthalein). According to the "acid control" theory, the sphincter should have remained patent as long as the duodenal contents in these experiments were alkaline. This it did not do, but was observed to close completely. However, absolute evidence that the amount of carbonate introduced neutralized all the acid in the duodenum could not be obtained, although but little doubt can exist but that neutralization was effected.

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[7] Luckhart, A. B., Phillips, H. T., and Carlson, A. J.: Contributions to the Physiology of the Stomach. LI. The Control of the Pylorus. *Amer. Jour. Physiol.*, Boston, 1919, L, 57.

CONCLUSIONS.

Tentative conclusions drawn from the experimental work here presented are as follows:

1. Finely divided carbohydrate, protein or fatty foodstuffs begin to leave the normal human stomach within from a few to 10 minutes after their initial ingestion.

2. Under normal conditions the human pyloric sphincter opens regularly at the approach of each antral peristaltic wave, allows chyme to pass through into the duodenum during an appreciable length of time, and closes when the antral peristaltic wave has spent itself.

3. The introduction of N/40, N/20 or N/10 HCl solutions into the first, second or third portions of the normal human duodenum either produced no effect on the opening of the pyloric sphincter, as observed by means of the fluoroscope, or produced effects which were interpreted as the result of abnormal irritation of the mucosa of the duodenum or sphincter.

4. The experimental results obtained do not support the theory that acid is the principal factor controlling the pyloric sphincter in man.

DISCUSSION.

DR. S. J. MELTZER, New York City: I should like, in the first place, to express a doubt whether this work was done in the Physiological Laboratory of Harvard.

DR. MCCLURE: It was done in the Peter Bent Brigham Hospital; but Dr. Cannon has been much interested in it and has gone over all the results very recently.

THE VALUE OF ESOPHAGOSCOPY IN THE DIAGNOSIS OF DISEASES OF THE ESOPHAGUS.

By Elmer B. Freeman, M. D.,

BALTIMORE, MD.

In this paper I desire to mention the indications for and the contra-indications to esophagoscopy and to discuss the value of this method of examination in the diagnosis of carcinomatous, spasmodic, cicatricial and compression stenosis of the esophagus.

The Indications for Esophagoscopy.

Any abnormal sensation referred to the region of the esophagus, any difficulty in swallowing or the presence of a foreign body should be sufficient evidence to warrant an esophagoscopic examination. In no other way may we hope to make an early diagnosis in many of the diseases of the esophagus. How fortunate it would be if, in cases of spasmodic stenosis at the level of the diaphragm, we could make the diagnosis before there was dilatation of the esophagus! I believe early esophagoscopy in these cases would reveal more frequently than we have thought a simple ulcer or an abrasion of the mucosa as the cause of the spasm. Esophagoscopy is essential in the diagnosis of stricture, not so much in the location of the stenosis as in differentiating between the various diseases causing the obstruction and in noting the condition of the esophagus above the stricture. In many cases of foreign bodies in the esophagus the diagnosis must be made by esophagoscopic examination, and in all cases their safe removal can be accomplished only by the esophagoscopic method.

The Contra-indications to Esophagoscopy.

The contra-indications to esophagoscopy are: aneurism of the thoracic aorta, cardiac disease with hypertrophy, arteriosclerosis with hypertension, thyroid disease, advanced pulmonary tuberculosis, cirrhosis of the liver and curvature of the spine in the cervical and dorsal regions. However, none of these are contra-indications if esophagoscopy is being done for the detection and removal of a foreign body.

STENOSIS OF THE ESOPHAGUS CAUSED BY CARCINOMA.

Cancer of the esophagus occurs most frequently in men. The early symptoms, as referred to the esophagus, are those of a mild dysphagia which gradually increases as the disease progresses, until, in some cases, symptoms of complete stenosis occur late in the disease. As a rule the location of the growth is at the anatomical narrowings of the esophagus. These are at the cricoid, the aortic arch, the left bronchus and the diaphragm. However, most of the growths occur in the middle third.

The esophagoscopic appearances of cancer vary greatly according to the stage in which the examination is made and also whether the growth is primarily in the esophagus or one of the adjacent viscera.

Jackson states that he has every reason to believe that the very early stage of cancer occurs as a leukoplakia in at least a few cases of cancer of the esophagus, but the opportunities for early esophagoscopy in cancer are so rare that there is no means of determining the frequency of such an onset. He states that in these cases the mucosa appears as though it had been burned with silver nitrate. In other instances an early manifestation of malignancy is submucosal infiltration, which gives a sensation of increased resistance in the esophageal wall. This is always the early finding when the carcinoma involves the esophagus from without, and in these cases the mucous membrane remains normal for a long time.

As the disease progresses other changes are noted, such as: irregularity in the lumen, the obliteration of the normal creases and folds, the absence of inspiratory and expiratory movements, and no wrinkling of the mucosa occurs if pressure is made with the esophagoscope.

Later on definite polypoid protrusions into the lumen with marked irregularity in the outline of the growth are noted. At this stage there is marked congestion of the mucous membrane and, in many cases, a beginning degeneration of the growth with ulceration. After ulceration has occurred the appearance of the ulcer is not entirely due to the carcinoma, but is partly due to a secondary infection with pyogenic organisms.

The ulcer is usually irregular in outline, the surrounding mucosa very much inflamed, the surface very granular, slightly elevated and bleeds easily when sponged. Definite areas of necrosis may be found in the more fungoid and cauliflower types.

Late in the disease one is impressed with the marked rigidity of the diseased area, the tortuosity of the lumen, the ease with which bleeding occurs, the absence of scar tissue and the small amount of dilatation above the growth. The small amount of dilatation is due to cancerous infiltration of the wall of the esophagus and to obstruction coming on late in the disease.

Endoscopically a malignant ulcer may be confused with one due to lues or tuberculosis. In luetic ulceration the following characteristics usually are seen: The edge of the ulcer is elevated and surrounded by markedly inflamed mucosa; the surface is frequently depressed; there is no excessive granulation tissue and there is very little bleeding when the ulcer is sponged, although the surrounding mucous membrane seems very much inflamed. The tuberculous ulcer is superficial; rather anemic in appearance; the granulation tissue is not excessive; the edges are not elevated; the surrounding mucous membrane is not markedly congested, and the surface does not bleed easily when sponged.

Before a differential diagnosis can be made, these findings, while very important, must be further corroborated by the clinical history, physical examination, X-ray and other laboratory studies. It is very easy, while making an esophagoscopic examination, to remove a specimen for microscopic study, and this should be done in all doubtful cases.

STENOSIS OF THE ESOPHAGUS CAUSED BY SPASM.

It is possible for spasmodic stenosis to occur at any point, but it is usually found where there is a definite anatomic narrowing, either at the cricoid or the level of the diaphragm. Normally there is a sphincter-like occlusion of the esophagus at the lower border of the cricoid, brought about by the lower fibers of the inferior constriction of the pharynx. A similar occlusion also occurs at the diaphragm, due to a sphincter-like arrangement of muscular fibers.

The spasm is usually reflex and may be caused by local diseases of the esophagus or associated with other diseases, the most frequent of which are irritative lesions of the gastro-intestinal tract. However, in many cases there is a basic neurosis.

Stenosis Occurring at the Lower Border of the Cricoid.—When the stenosis occurs at the cricoid the disease is essentially a spasm of the circular fibers of the inferior constriction of the pharynx, the presenting symptom of which is difficulty in swallowing. This difficulty the patient describes as inability to start the bolus of food downward, but after the food is once started it passes uninterruptedly into the stomach. There is no regurgitation of food except in those cases where there is an associated spasm at the diaphragm. A dysphagia of this sort denotes a high obstruction of the esophagus, the character of which must be determined by an esophagoscopic examination. In a few cases the spasmodic stenosis will be found to be due to an organic lesion.

Some cases of spasmodic stenosis occurring at the cricoid cannot be endoscopically differentiated from the spasm that always occurs upon introducing the esophagoscope. In other instances the mucosa is apparently thrown into folds which radiate from a central point and, in other cases the entrance into the esophagus appears as a transverse or crescent-like slit. The mucous membrane is normal in appearance except in those cases in which an organic lesion is the cause of the spasmodic stenosis. In such cases the appearance of the mucosa will depend on the organic lesion present.

By far the most important endoscopic finding is that when slight pressure is made with the esophagoscope the spasm relaxes and no further difficulty is experienced in passing the instrument through the esophagus. The same relaxation may be produced by having the patient breathe deeply. I desire to report the following case:

Case Report: Mrs. J. W., a white woman, age 30, entered the Maryland General Hospital March 10, 1920.

Complaint.—Dysphagia; fullness and discomfort after eating.

Family History.—Negative except that mother died, age 42, of carcinoma of the stomach.

Past History.—Negative for infectious diseases, but the patient has complained of the following digestive symptoms for ten years: fullness after meals, some nausea, occasional vomiting, marked eructations of gas and obstinate constipation.

Present Illness.—For six months prior to entering the hospital patient suffered from dysphagia which she described as inability to swallow her food. Only about half of her attempts at swallowing were successful and it would take her an hour to eat a meal that a normal individual would eat in thirty minutes.

Physical Examination.—Rather pale, anemic looking woman, showing evidence of marked loss in weight; pupils react to light and accommodation; no tenderness over the sinuses, antrums or mastoids; slight purulent discharge from the left ear; teeth present definite evidence of pyorrhea; throat negative; heart of normal size; sounds clear at the base and apex; blood pressure, 136/86; lungs apparently normal to percussion and auscultation; abdomen: narrow costal angle, muscles relaxed, freely movable right kidney and no local tenderness; extremities normal; reflexes active.

Laboratory Studies.—Urine analysis, normal.

Stomach Analysis.—Fasting stomach. No seven-hour retention. Test meal, free HCl absent; total acidity 14°; mucus, two plus.

Stool Examination.—Occult blood absent.

Blood Examination.—Hemoglobin 90 per cent.; erythrocytes, per c.mm., 4,736,000; leucocytes, per c.mm., 7,600.

Fluoroscopic Examination.—Chest. Very small heart of vertical type; no dilatation of the aorta; apices of the lungs apparently clear; no irregularity of the diaphragm. Esophagus—no esophageal dilatation. Stomach—fish-hook type; four fingers below the crest; well to the left; very atonic; good duodenal cap brought out by palpation; no cecal stasis; hepatic flexure below the crest; transverse colon prolapsed well into the pelvis; very spastic.

Esophagoscopic Examination.—This revealed a definite spasmodic stenosis at the cricoid region.

(*Note.*—The spasm was relieved by the passage of the esophagoscope and dilatation with a No. 40, French scale, bougie every third day for four weeks.)

Stenosis Occurring at the Diaphragm.—Usually in these cases there is no difficulty with the bolus of food entering the esophagus, but the difficulty is with the food entering the stomach. These patients generally have the sensation of the food being lodged behind the lower end of the sternum, the sensation being described as fullness without pain. Later in the disease many symptoms are added which are due to the dilatation of the esophagus and disturbances in nutrition.

In cases of spasmodic stenosis at the diaphragm no difficulty is experienced in introducing the esophagoscope until it reaches the level of the diaphragm. There it meets definite resistance, but with gentle pressure the spasm relaxes and the esophagoscope passes into the stomach. Early in the disease the endoscopic appearance of the esophagus may not differ from the normal. Late in the disease the esophagus is very much dilated, the walls are atonic, the mucosal folds obliterated, the respiratory movements are absent, the mucous membrane shows evidence of chronic inflammation and in many cases superficial ulceration is present. Personally, I believe that at least a few of these cases have their origin in ulceration of the esophagus. The condition I have described above is usually diagnosed as chronic cardiospasm. However, in my experience I have never seen a spasmodic stenosis at the cardia and I agree with Jackson that the spasm occurs at the level of the diaphragm.

Case Report: Mr. J. S., age 42, carpenter, admitted to Out-Patient Department of Johns Hopkins Hospital, April 15, 1914.

Complaint.—Difficulty in swallowing.

Family History.—Negative.

Past History.—Negative.

Present Illness.—For six years previous to coming under observation patient complained of difficulty in swallowing. He stated that the food seemed to lodge behind the lower end of the sternum. This was followed by regurgitation of food and the patient discovered that by drinking one or two glasses of water after eating, a portion of the meal entered the stomach and the regurgitation was relieved. He had lost about forty pounds in weight and had been unable to work at his trade for two years.

Physical Examination.—Negative for organic lesions.

Laboratory Studies.—Blood: Wassermann reaction negative.

Urine Analysis.—Negative.

Stool Examination.—Occult blood negative.

Stomach Analysis.—Unable to introduce tube into the stomach, but obtained approximately 1,000 c.c. of a foul smelling fluid from the esophagus that contained a large amount of mucus and undigested food.

Fluoroscopic Examination.—Esophagus very markedly dilated with definite obstruction at the level of the diaphragm; bismuth passed into the stomach very slowly. Stomach normal in size and below the crest; no deformity of duodenal cap; no cecal stasis; hepatic flexure below the crest; transverse colon prolapsed into true pelvis.

Esophagoscopic Examination.—Esophagoscope passed without difficulty to level of diaphragm and there definite resistance was met, but gentle pressure caused the spasm to relax and the instrument passed into the stomach. There was a small scar at the level of the diaphragm and the esophageal wall was very atonic, a dirty gray in color, chronically inflamed and presented several small superficial ulcers.

(*Note.*—The stricture was dilated with a Plummer dilator twice a week for ten weeks, after which time he was apparently cured, but a fluoroscopic examination showed that there was no decrease in the size of the dilatation and that food passed into the stomach slower than normally. This patient returned to work two months after treatment was discontinued and has been symptomatically relieved since that time. The dilatation has persisted.)

Stenosis of the Esophagus Caused by Cicatricial Tissue.

Cicatricial stenosis is the result of a healed ulcer, either simple, tuberculous, leutic or associated with one of the acute infectious diseases. A simple ulcer is usually traumatic and is generally caused by the swallowing of a corrosive liquid or the lodgment of a foreign body in the esophagus. However, some are due to less violent trauma and may result from slight abrasions caused by the swallowing of coarse articles of food.

The symptoms complained of, as referred to the esophagus, depend almost entirely on the degree of stenosis. In many cases with slight stenosis no dysphagia is noticed until the patient tries to swallow some coarse article of food, while in the cases with marked stenosis the patient is unable to even swallow liquids. Symptoms of complete stenosis may occur in these cases due to the lodging of some coarse article of food in the stricture.

Stenosis Caused by the Swallowing of a Corrosive Liquid.—The stricture which results from the healing of an ulcer caused by the swallowing of a corrosive liquid may occur at any point in the esophagus, but it usually occurs at one of the anatomic narrowings. In my experience this form of stenosis occurs most frequently in the middle third at the crossing of the left bronchus; next in frequency in the lower third at the level of the diaphragm, and as yet I have not seen a case in the upper third of the esophagus. The degree of stenosis varies from a slight narrowing to a complete occlusion of the lumen. In some cases there may be more than one stricture.

The appearance of the esophagus depends on the degree of stenosis, the duration of the disease and the character of food taken by the patient. If the stenosis is slight and the esophagus has not been irritated by coarse food, the mucosa appears normal and there is no dilatation above the stricture. If the stenosis is of high degree, marked changes in the wall of the esophagus will be noticed above the stricture and all food and secretion must be removed before one can make a satisfactory examination. Cicatricial tissue in the esophagus is always paler than the normal mucosa and may appear almost white. The appearance of the stricture depends upon the location of the scar. In some cases a linear scar will be seen with a drawing in of the wall of the esophagus. In other instances the scar is seen to cross one side and cause a flattening of the lumen. In others the scar causes a polypoid protrusion into the lumen, and in many cases the scar tissue almost encircles the esophagus, causing an anular stricture. In the high grades of stenosis definite dilatation of the esophagus occurs above the stricture. The mucosa is inflamed and frequently there are noted superficial abrasions and sometimes ulceration. This condition of the mucosa above the stricture no doubt increases the symptoms of obstruction by reflexly causing the spasm.

Case Report: Master H. R., a child, three years old, was referred to me by Dr. Bloodgood and Dr. Crist.

Clinical Note.—Entered St. Agnes' Hospital September 15, 1916, with the following history: One year ago drank some concentrated lye which caused a gradually developing stricture that could not be relieved by frequent attempts at dilatation and at the end of six months a gastrostomy was performed to save the child from starvation. About five months after this operation, Dr. Crist succeeded in passing a "uterine sound from the stomach upward," after which the child was able to swallow liquids for three weeks. When I first saw him, one year after the drinking of the corrosive, he was emaciated, anemic, and markedly prostrated. Radiographic examination showed an apparently complete stenosis at the crossing of the left bronchus with marked dilatation above the stricture. The esophagoscopic examination confirmed the radiographic diagnosis of stricture with dilatation. It further revealed a markedly inflamed mucosa with a number of superficial abrasions. After removing some particles of food and secretion from the stricture it was found that the lumen was not completely occluded and after a number of attempts, we were successful in passing a No. 12, French scale, bougie.

Stenosis Following the Removal of Foreign Bodies.—The stricture which results from the healing of an ulcer caused by the lodgment of a foreign body most frequently occurs in the upper third, which is the narrowest part of the esophagus. Some may occur in the middle and lower third.

Recently I saw a patient who complained of pain and difficulty in swallowing and stated that Dr. Johnston, of Baltimore, had removed a foreign body from the esophagus two years previously. A esophagoscopic examination revealed a moderate degree of stenosis in the middle third.

Stenosis Due to Lues.—Cicatricial stenosis of the esophagus due to lues is rare. However, dysphagia coming on after middle life makes one think of syphilis as a possible cause of the stenosis. The stricture most frequently occurs in the upper third. Endoscopically the cicatrix due to syphilis does not differ in appearance from scars due to other causes. I wish to report the following case of stenosis due to lues.

CASE REPORT: Mr. J. L., age 40, white, married, referred July, 1913, by Dr. Hobbleman.

Complaint.—Dysphagia.

Family History.—Negative.

Past History.—Typhoid at the age of 34; lues ten years ago. Moderate user of alcoholic stimulants for 15 years.

Present Illness.—Began to suffer about six months ago with "pain on swallowing," which was soon followed by a dysphagia that became progressively worse and when I first saw him he was able to swallow only small quantities of liquids. He also complained of dyspnea, marked prostration and rapid loss of weight.

Physical Examination.—Upon examination the patient looked very ill, was emaciated and slightly cyanotic. Otherwise the general physical examination was negative.

Laboratory Studies.—Our routine laboratory studies revealed a three plus Wassermann reaction as the only positive finding of interest.

Esophagoscopic Examination.—This showed a dense scar involving the upper end of the esophagus and the wall of the pharynx.

Stenosis Due to Tuberculosis.—Cicatricial stenosis of the esophagus due to tuberculosis is said to be extremely rare. However, it might be found to be more frequent if a routine esophagoscopic examination was made in tuberculous patients suffering with dysphagia. This is illustrated by the following case:

CASE REPORT: Mrs. I. C. L., age 30, white.

Clinical Note.—Admitted to St. Agnes' Hospital and assigned to the Medical Service, where a diagnosis of pulmonary and laryngeal tuberculosis was made. She complained of marked difficulty in swallowing. An esophagoscopic examination was made and it revealed a stenosis of the upper end of the esophagus, resulting from an extension of the tuberculous process in the larynx.

Stenosis Associated with Acute Infectious Diseases.—Cicatricial stenosis due to the healing of ulcers associated with the acute infectious diseases has been well established, but it has not come under my observation.

STENOSIS OF THE ESOPHAGUS CAUSED BY COMPRESSION.

Compression stenosis of the esophagus may be due to an enlarged thyroid, cardiac hypertrophy, or aneurism. However, it is usually due to mediastinal disease. Endoscopically the lumen of the esophagus at the site of the compression appears as a transverse slit and the normal folds are obliterated. The mucosa is normal in appearance except in a few cases where there is a chronic inflammation due to stasis.

If, while making an esophagoscopic examination, definite resistance is met and the wall of the esophagus is apparently normal, one should always bear in mind the probability of a compression stenosis. In these cases no attempt should be made to pass the obstruction. Compression stenosis can usually be diagnosed by other methods and esophagoscopy should not be done in these cases except for the detection and removal of foreign bodies.

CONCLUSION.

In conclusion I wish to warn against esophagoscopy in compression stenosis, to emphasize its value in the diagnosis of cicatricial and spasmodic stenosis, and, finally, to urge esophagoscopy as a routine examination in all cases with symptoms referred to the region of the esophagus, so as to make possible the early diagnosis in many of the diseases of the esophagus.

DISCUSSION.

(See page 154.)

DIAGNOSIS AND CLINICAL MANIFESTATIONS OF CARDIOSPASM ASSOCIATED WITH DIFFUSE DILATATION OF THE ESOPHAGUS.

By Frank Smithies, M. D.,

CHICAGO, ILL.

1. General Consideration.

Definition.—A condition in which there occur diffuse dilatation of the esophagus with maintenance of the normal contour of the gullet, in association with a varying degree of unusually strong and prolonged contraction or hypertrophy of the cardiac sphincter. There may co-exist hypertrophy of the muscle layers of the lower esophageal wall. The hypertrophy of the cardiac sphincter may be of but moderate extent, but when it is complicated by powerful spasmodic or long continued contraction, there results transient or long maintained, frequently complete, obstruction to the passage of esophageal contents from the gullet into the stomach. Again, the hypertrophy of the cardiac sphincter may be advanced, and then such hypertrophy may of *itself* result in functional stenosis of the cardia, whether or not there is the complication of associated spasm. In marked instances of the affection, the hypertrophy of the cardiac sphincter may resemble in degree such hypertrophy as is found at the pyloric sphincter in patients affected with the hypertrophic stenosis of infancy. Cases of "cardiospasm" described where muscular hypertrophy of the cardiac sphincter could be not demonstrated at autopsy or operation would seem to be errors in diagnosis or faults in the observation of what actually constitutes muscular hypertrophy in a dilated, enlarged, static esophagus. There have been too few studies of the normal cardiac sphincter to permit comparison. During the last few years, many papers have appeared purporting to describe "cardiospasm," but in truth they have dealt with instances of *spasm* at the *cardia* or of transient esophageal spasm, local or general.

Nomenclature.—It should be definitely understood that the terms "spasm at the cardia" and "cardiospasm" are not synonymous. Only the latter form of affection includes hypertrophy of the cardiac sphincter, or the lower portion of the esophagus, combined with general diffuse dilatation of the esophagus. "Spasm at the cardia" is a transient lesion of itself, and it is not associated with uniform, diffuse dilatation of the esophagus, until it has been so long existent, or is of such constant maintenance, as to produce esophageal changes not of a transient nature, particularly an associated, permanent dilatation of the gullet.

Etiology.—In cardiospasm, the dilatation of the esophagus would appear to be due to rather indefinitely understood causes. Rosenheim has suggested as an explanation, primary atony of the muscular coats of the gullet; Kraus considers the condition due to the development of persistent, long maintained spasm of the cardia, associated with paralysis of the circular muscles of the esophagus, the latter occurring as a consequence of degenerative changes in the vagi. Numerous authors, as, for example, Fleiner, Zenker and Sievers, maintain that dilatation results from a congenital predisposition on the part of the esophagus; Martin considers a primary esophagitis as an important factor. In addition, there have been advanced other explanations, viz.: kinking of the esophagus at the cardiac opening; anatomic defects associated with the esophagus or the stomach, such as ulcer or carcinoma; congenital or acquired asthenia. Plummer, following Mikulicz and Meltzer, is inclined to the opinion that, in addition to the changes occurring at the cardiac sphincter, there exists some disturbance of the nerve-muscle mechanism of the esophagus, which may permit diffuse dilatation of the gullet, irrespective of actual stenosis due to hypertrophy of the cardiac sphincter. Plummer emphasizes that in some patients, there may be noted the occurrence of a *habit of spasm* at the cardia, and this without anatomic diffuse dilatation of the esophagus. Numerous such instances have found their way into the literature, misnamed "cardiospasm."

In my opinion it is quite likely that such extra-esophageal lesions as give rise to repeated, powerful, long-maintained contractions at the cardia are capable of producing the muscular hypertrophy of overwork; coincidentally, this alteration in sphincter reflex time secondarily disturbs the normal neuro-muscular coordination in the esophagus above it. As a result, the overworked sphincter seems to develop its own independent contraction rate, and this of a duration beyond the normal time limits, and out of the normal rhythm of the swallowing act. In such circumstances the stimuli from above (formerly passing down to a cardiac sphincter, normally contracting and relaxing in appropriate time and with limited degree) are then delivered in an uncoordinated fashion and at irregular time intervals with little or no effect upon the systole and diastole of the cardiac sphincter. The initial overstimulation to the cardia would seem to be exerted by numerous anomalies. Such may be extra-esophageal or intrinsically esophageal. These stimuli may arise from the central nervous system, from the esophagus itself, from the diaphragm, the intrathoracic organs, from the stomach, duodenum, gall-bladder, appendix or other abdominal viscera. In some patients it would seem that more than usually powerful cardiac contractions are initiated by psychic upsets. However, it should be emphasized that cardiospasm is by no means an affection only of psychesthenic or of neurologically unstable individuals. While the initial cause of spasm at the cardia may produce a more active response in individuals who are psychically, or nervously, abnormally responsive to *all* stimuli, it should be recognized that in well-developed instances of cardiospasm, the lesion which has to be dealt with is an anatomic one and not a mere functional disturbance. In these patients, in spite of the opinion to the contrary held by some clinicians, I believe that there always exists a definite hypertrophy of the muscular cardiac ring (such as can be shown when skilled fluoroscopic study is made during the taking of food). With this is always readily demonstrable (although of varying degree) generalized dilatation of the esophagus. It is important that these facts be recognized, because, upon their realization, depends the exhibition of proper methods of treatment. Patients affected with this lesion require more accurate therapeutic procedures than rest cures, cessation from mental, psychic or physical over-activity, quieting medicines, christian science, osteopathic hocus-pocus and the like.

Structural Changes in the Esophagus.—The anatomic anomalies present in cardiospasm should be emphasized, viz.: hypertrophy of the cardiac sphincter, atypic cardiac contractures (powerfully spasmodic or abnormally persistent) and diffuse enlargement of the esophagus without local or general gross alterations of its contour. The *effect* of these anatomic changes is to cause dysphagia, imperfect passage of foods into the stomach and generally some degree of permanent esophageal retention. *The quantity of re-*

tained food varies widely in different patients and in a given patient at various times during the study of his affection. The esophagus may retain from less than an ounce to as much as several liters of fluid or mixed food. Upon the patient *systemic effects* are those consequent upon slow or rapid starvation. The degree of malnutrition may be so pronounced and of such acute development as to suggest malignant disease or advanced cicatricial stenosis caused by peptic, luetic, tuberculous or traumatic ulcer of the gullet.

II. Material Studied.

During the past seven years, 76 instances of cardiospasm associated with diffuse dilatation of the esophagus have come under observation. Tabulated records of 47 of these cases are available for study.

Sex.—Of the 47 cases there were 25 females and 22 males. This approximate equality of the sex ratio is rather striking in view of the prevalent opinion among practitioners that females are much more apt to be affected.

Age.—The average age for the series was 39.2 years; the youngest case was age 19 and the oldest 70. It is thus seen that true cardiospasm is a disease common to mid-adult life, the majority of instances occurring between 25 and 35.

Occupation appears to play a very small part as an etiologic factor, but the ailment seems to be relatively common in those individuals who are very active, physically and mentally. Temperament has been rather unduly emphasized in its relation to the initiation of cardiospasm. While it is true that some of these patients are delicately balanced psychically, yet a goodly number is of what one would call a stable temperament.

Ailments previous to the onset of cardiospasm with a few exceptions appear to have little bearing with regards to causing the affection. It was noticed in our series that in eight instances, frequently recurring attacks of bronchitis or asthma were associated with dyspepsia and ultimately with dysphagia. These severe coughing spells might be a not unimportant factor in causing cardiac irritation and neuro-muscular fatigue of the diaphragm. There were three instances of gastric ulcer proved by laparotomy, but the cardiospasm was in no way influenced by the operative procedure upon the stomach. In but seven instances did shock, fright or acute fatigue precipitate the symptoms (two were neuroses). Excessive smoking, particularly of cigarettes, was observed in five cases. This might offer an interesting etiologic suggestion to those who attribute cardiospasm to vagus malfunction.

Mode of Onset.—In but 17 instances the affection with the associated dysphagia was acute in its inception (36 per cent.). In 30 cases (74 per cent.), the affection appeared gradually and was not infrequently punctuated by attacks of exaggerated distress.

Duration of All Symptoms.—This averaged for the series 5.6 years and ranged for the individual cases, from three months to 20 years. The degree of dysphagia or esophageal retention, not infrequently bore no relation whatever to the time that symptoms had been present. Not rarely esophageal retention of as great as 1,000 c.c. arose in individuals who had been ill for less than a year (nine cases).

The duration of the presenting trouble averaged for the series 4.2 years with a range in the individual cases of from two months to 20 years.

The Dysphagia is not commonly painful. Usually there is a sensation of fullness or tension beneath the sternum, a feeling of tightness or a discomfort often described as "crowding of the heart." There may be dyspnea or cough. The first swallows of food may produce the uncomfortable sensation. It is of diagnostic significance to know that liquid foods are more prone to immediately bring about distress than are solids. In this way, the type of dysphagia differs strikingly from that common to other forms of anatomic stenosis (ulcer, cancer, trauma, etc.) along the course of the esophagus. There are instances, however, where the swallowing act at once excites sharp cramp-like pain, usually located beneath the xyphoid, or along the spine, from the 9th to the 12th dorsal vertebrae. The pain may be at first sufficiently acute to cause incapacitation, and then may be followed by intermittent or continuous gripping, binding, or sticking type of discomfort.

In our series of patients there was constant dysphagia in 45 cases. There was dysphagia to fluids only in 21, to solids only in 14, and to all foods in 12 cases.

Dysphagia is nearly always accompanied by *vomiting or spas-*

modic food regurgitation so long as esophageal muscle-tone remains good. A fairly characteristic and differential feature of the vomiting is *that it is usually sudden, frequently explosive and very often occurs shortly after the ingestion of food.* Liquids are more apt to produce sudden, copious emesis than are solids; in fact, the ingestion of soup or fluid at the early part of the meal may bring about spasmodic emesis of such sudden and forceful nature that the individual is compelled to rush hastily from the table to avoid accidents. This prompt vomiting of liquids is frequently aggravated by the fact that but rarely in general practice is the lesion of cardiospasm recognized. To the average physician a dysphagia presumably calls for a liquid diet and he accordingly orders such. Solids or partly solid foods are generally well borne, particularly in the early stages of cardiospasm. The constant attempt to take liquids is followed promptly by vomiting, lack of nourishment and malnutrition and doubtless added damage to the weakened esophageal wall. The patient frequently finds this out himself and refuses to follow physician's instructions, when liquid diet has been urged. In advanced cardiospasm, practically all the food taken may be vomited within an hour or two following its ingestion, or, if dilatation of the esophagus is pronounced, morning retention vomiting is common and constant.

The *vomitus* consists commonly of foods whose appearance resembles that of food as eaten. It is alkaline in reaction, usually contains much mucus, rarely lactic acid or blood and may have a high bacterial content. At times vomiting is delayed and only when a large meal is taken at night is emesis produced. At that time, food eaten at breakfast or even the night before may be vomited unchanged. Sometimes the vomitus is copious at night and prevents proper rest and sleep. A few cases of even marked cardiospasm do not manifest actual vomiting, but there is constant belching, bad taste in the mouth, eructations of food and frequently copious regurgitation of ill-tasting fluid, food or mucus. Sometimes *nausea* is pronounced and there occurs *remarkable increase in the secretion of saliva.* This may indicate central irritation of the salivary glands. Even when some food passes the cardia, the regurgitation of frothy, thick, tenacious mucoid material is apt to be annoying, particularly when such occurs toward evening. Consequent upon the vomiting, appear evidences of lack of fluid in the tissues, constipation, diminution of urine output, headaches, dizziness and anemia.

In this series of cardiospasm patients 31 vomited daily (frequently many times during the day.) In 22 cases retention vomiting was observed. Frequently, food (fruit skins, pieces of tomato, meat, lettuce, milk clots, etc.) was retained in the lower part of the dilated esophagus for as long as a week. In 17 of our patients the vomiting was projectile. In such cases it was a common experience that patients began vomiting as soon as food was swallowed. There was vomiting of blood in 14 cases. This hematemesis, seemingly, bore relationship to dilated or varicose veins in the lower esophagus, although one could not always be certain that ulceration had not taken place in some portion of the esophageal wall where varicosities were not present. In one case the hematemesis appeared to be definitely vicarious, inasmuch as it occurred on two occasions just before menstruation.

Regurgitation, without actual vomiting, was especially annoying in 15 patients. This regurgitation was generally uncontrollable, the regurgitant food mass was frequently very copious in quantity and of foul, sickening odor. There were patients in which periods of intermittent regurgitation preceded cycles of exhausting vomiting.

Miscellaneous Clinical Symptoms.—Weight loss was experienced by all our patients. It averaged for the series, 28.2 pounds. In individual cases it ranged from ten to as much as 82 pounds. The appetite was stated as being lessened or as being poor in 64. Nine and one-half per cent. of patients experienced pronounced anorexia. Normal bowel movements were recorded in 47 per cent. of the patients. In the remainder there was commonly obstinate constipation. This constipation seemed to be due largely to insufficient quantities of food or fluid passing the cardia into the stomach and bowel.

III. SPECIAL DIAGNOSTIC MANEUVERS.

In true cardiospasm an unguided *stomach-tube,* passed into the esophagus, usually excites free food regurgitation about the tube, and the spasmodic emesis of much food and liquid through the tube. The tube may be passed usually freely as far as the normal cardia, but there it meets an elastic resistance. This resistance is occasioned either by the tube impinging on the distended esophageal wall and depressing its lower sixth, into a sac-like pouch, or by pronounced and persistent contracture of the cardiac sphincter. In but a few not well established instances of the affection does the cardia lie in a line direct from the pharynx and thus allow an unguided stomach tube to be passed to it, and then by exertion of pressure, admit of the cardia's being forced and the tube being carried into the stomach. If the tube is guided upon a silk cord to the cardia (as suggested by Mixter of Boston 17 years ago and emphasized by Henry Plummer clinically), the tube can be carried direct to the cardia and, provided the tube is a stiff one, can, with exertion of a moderate degree of force, be passed into the stomach. The "giving way" of the tight, elastic cardia can be felt as the stomach-tube passes the hypertrophied or permanently contracted sphincter.

If not guided to the cardiac orifice by a cord, a *bougie* passed into the esophagus traverses the lumen further than the average distance of the cardia from the incisor teeth. Usually the olive may be passed considerably further than normally and the unsuspecting manipulator may fancy that he is well beyond the cardia and that the olive lies in the stomach, it having passed the cardiac sphincter without the operator's knowledge. This mistake occurs as a result of the bougie passing downward and to the right in the folds of the sac-like, pendulous, lower esophagus. In such circumstances, forcible pressure is dangerous; in our own experience, one case of esophageal rupture occurred after the patient had been elsewhere "dilated by olives" for six months and the cardia was later devulsively dilated. At the autopsy it was found that at the lowest point of the dilated esophageal sac, and about two inches from the cardia, there were denudation of the mucous membrane, marked thinning of the wall of the esophagus and peri-esophageal inflammatory thickening. The area of the esophageal attenuation was about the size of a large olive, somewhat depressed, and lay in a straight line from the pharynx to the lowest part of the esophageal sac, but was out of the line from pharynx to cardia. In this patient, a devulsing dilator, guided upon a silk cord, passed freely into the cardiac orifice, but the increase in intraesophageal pressure during dilatation was sufficient to cause rupture of the esophagus at the spot where repeated bouginage had weakened the walls.

Even in long established, obstinate cardiospasm, it is generally possible for a *silk cord guide* to pass from the esophagus into the stomach, but at times the cord may be held in the esophagus several days and may pass freely only when spasmodic contracture of the cardia has been minimized by the free use of atropin or of belladonna. However, unless the dilated esophagus be washed clean of retention contents, silk cords may lie for weeks without passing from the gullet to the stomach.

An olive bougie of large size passes readily to the cardia upon a taut string; moderate pressure "forces" the sphincter and permits the bougie passing into the stomach. Withdrawal of the bougie may be accompanied by a readily recognizable, elastic resistance. It is usually the experience that the bougie exerts little influence with respect to permanent enlargement of the cardia opening or permanent relief of the dysphagia. It is quite as sensible to expect permanent dilatation of a cardia in cardiospasm by bouginage, as it is to expect a spasm of the anal sphincter will be relieved by frequent digital examinations. If the patient be examined at different times of the day or upon different days, a variation in the amount of resistance at the cardia may be determined, but it is characteristic of the affection that at every examination there is resistance present.

The Roentgen examination is of great aid in diagnosis, but the evidence which it supplies is not infallible. Fluoroscopic observation and plate studies must be carefully interpreted in the light of clinical and physical examination and exploration of the esophagus by guided instruments. Fluoroscopically, when the patient swallows the opaque meal, it will be seen that usually a small amount of the mixture immediately passes through the cardia into the stomach. In moderately well-established instances of the affection, the cardia then closes quickly, with or without several easily recognized squirty contractions and continued ingestion of the opaque mixture results only in filling out the esophagus; practically nothing passes into the stomach. It can then be shown that the esophagus is uniformly dilated, that its wall is regular, that dilatation occurs along anatomic lines and that the cardiac end of the esophagus down to the hypertrophied sphincter presents no gross irregularities of contour. In shape, such a dilated esophagus resembles, characteristically, a large lily-of-the-valley leaf or is roughly pear-shaped. The dilatation may be enormous and extend so markedly throughout the entire length of the esophagus as to produce pressure upon the heart, the aorta or the trachea. The obstruction at the cardia may be absolute and may be maintained for hours or days, until emesis or lavage results in an apparently reflex, partial relaxation of the hypertrophied cardiac sphincter. Again, in moderately advanced cases, esophageal retention may persist from a half to two hours and then the opaque meal passes in a jerky, spasmodic fashion, slowly through to the stomach. Even in these cases, however, one rarely finds that the esophagus completely empties itself within from two to six hours. In the early incompletely established cases, esophageal retention may be but transient. Fluoroscopically, it is possible to observe alternate, spasmodic contractions and relaxations of the cardiac sphincter, associated with the intermittent passage of small amounts of the opaque meal from the lower esophagus into the stomach. The esophagus will thus empty in from a few minutes to an hour. Mild instances of the affection are not generally associated with great sac-like esophageal dilatation.

In rare instances, fluoroscopy and Roentgen plates fail to make the diagnosis; the Roentgen findings seem characteristic for cardiospasm, but in the dilatation of the esophageal sac, ulceration, diffuse scirrhus carcinoma or lues remain masked even until they have advanced sufficiently to produce extensive stricture or to cause death from metastasis. Not rarely, fluoroscopy clearly demonstrates torsion of the terminal esophagus with or without rather sharp angulation or displacement usually to the left of the midline of the thorax. In several instances of well developed cardiospasm with diffuse dilatation of the esophagus, I have noted unusual "doming" of the diaphragm. In these patients the diaphragm movements were atypic: degree of depression was unequal on the two sides, rates of downward movements were not equal on the two sides, *i. e.*, the diaphragm depressions were not synchronous and, locally, in the region of the hiatus, there seemed to be a sort of "piling up" of diaphragm (local hypertrophy?). Observation of the above phenomena has suggested to me that, at times, the fault primary in initiating cardiospasm lies in the muscle or serous layers of the diaphragm: inflammatory processes in the serous covering of the esophagus, lungs, heart, diaphragm or a local myositis of the diaphragm itself, with resultant disturbance in the neuro-muscular mechanism of the diaphragm, and, subsequently, of adjacent viscera with their correlated nerve supply.

It should be scarcely necessary to mention that fluoroscopy, conducted with the patient only in the anterior-posterior position, can, at its best, give but incomplete information. Unless the patient is examined also laterally so that the course of and the anomalies in the esophagus are susceptible of complete exposure, much of interest and of clinical usefulness will be lost. It is advisable to examine fluoroscopically, during the swallowing act with the diaphragm depressed by deep inspiration, the breath being held and the diaphragm fixed during swallowing. The patient may be turned rapidly from the anterior-posterior to the lateral ("quartering") position and by this manouvre one may secure views of the lowest portion of the esophagus, of the sphincter, the contour, peristaltic activity and the position and shape of the diaphragm. For careful scrutiny of the terminal esophagus and the cardiac sphincter, the diaphragm of the fluoroscope should be closed to such point as will permit minute study of the esophageal contour inch by inch. Such procedure is necessary to detect or to rule out ulcers, scirrhus carcinoma, etc., and to properly study the sphincter and its action. The successive employment of thin fluid and thick mush-like opaque suspensions, gives useful information relative to sphincter action, esophageal peristalsis, stasis, contour and degree of dilatation. The patient should be examined at least on two successive days. The second study should be carried on after the exhibition of antispasmodic medicines to physiologic tolerance. Only in this way can one determine the true condition existing at the cardia and the actual size of the esophagus. If this procedure were routinely followed, many "literature" cardiospasms would disappear—for many such are only "spasm at the cardia."

Roentgenograms should be made with the patient in the semilateral or "quartering" position as well as the anterior-posterior. When plates are made with the patient in the semi-lateral position, the entire esophageal contour may be delimited. In order to bring out the cardia and the lower portion of the esophagus it is advisable to have the patient drink as much of the opaque meal as he is able, then clasp his hands high above his head and take a deep breath. The inspiration depresses the diaphragm and generally enables accurate visualization of the lower esophagus. The best roentgenograms are made with the patient standing with his left side next to the plate. When extensive esophageal dilatation is present, anterior-posterior roentgenograms also should be made in order to determine the lateral limit of the esophageal sac and to demonstrate the degree of encroachment of the enlarged esophagus upon the heart, aorta and lungs. Such plates frequently demonstrate that the lowest point limit of the dilated esophagus lies several inches below the level of the cardiac sphincter. This shows vividly how futile is the effort to pass bougies or tubes into the stomach without their being guided from pharynx to cardia by a swallowed silk thread. In patients where the cardiospasm is not far advanced, anterior-posterior roentgenograms even may fail to indicate that the condition is present. In such circumstances the esophageal shadow is overlapped and hidden by the shadows of the heart, the aorta and the spine. Series of plates should be made before and after the administration of antispasmodic medicines (belladonna, benzylbenzoate, etc.). Such permit of interesting comparative studies and furnish valuable records of anatomic changes as the function of the esophagus is restored by treatment.

Esophagoscopy.—Inasmuch as in certain cases of cardiospasm the initiating fault lies in an esophageal lesion, it would appear a proper procedure to explore by sight the entire esophageal mucosa from pharynx to cardia, that is, provided that one who is familiar with esophagoscopy and who knows the significance of what he sees is able to do the work. Much esophagoscopic work as done is worthless because the operator knows little of the appearance of the normal esophagus. In well-established instances of cardiospasm, an actual view of the interior of an enormously dilated gullet and an anomalously acting sphincter is truly worth while, if for no other reason than to satisfy one's curiosity. Where some doubt exists respecting whether or no a cardiospasm state is complicated by scirrhus cancer, peptic ulcer, foreign body, gumma or extra-esophageal ailment, certainly esophagoscopy is a justifiable procedure in clearing up doubtful points in diagnosis even though it is a bit annoying to the subject. A thoroughly cleansed esophagus, carefully administered in intratracheal anesthesia and an esophagoscope, such as the particularly ingenious Sussmann, greatly facilitate the procedure. When powerful contractures of the cardiac sphincter persistently recur, following most expert divulsive treatment, it would seem practically necessary always to perform esophagoscopy with the object of searching for and treating locally, irritative mucosal lesions.

Prognosis.—If the affection is properly managed, there is clinical and functional recovery in about 70 per cent. of instances of even well-established instances of cardiospasm. Improvement occurs in 20 per cent. About 10 per cent. of patients are not permanently benefited by any form of treatment at the most expert hands. The lesion seemingly resists all forms of therapy. In this class are patients extremely emaciated, instances of irritable esophagus where proper treatment cannot be carried out and very likely cases where there is a congenital defect or, as Plummer has emphasized, the persistence of a "habit" of spasm. For these unfortunates, gastrostomy may be required to save or prolong life. If, with cardiospasm there are such associated lesions, as gastric ulcer or malignancy, gallstones, appendix infection or ulceration or inflammation of the cardia or esophagus itself, prognosis is seriously limited unless these lesions are amenable to appropriate medical or surgical treatment. Even after restoration of the cardiac sphincter action, the esophagus may remain much dilated and permit food retention. However, it is surprising how great dilatation of the esophagus may persist and yet such is unaccompanied by troublesome dysphagia, provied the cardiac orifice is patent and the sphincter acts normally.

DISCUSSION.

(See page 154.)

ROENTGENOLOGY OF THE ESOPHAGUS.

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My part in the program will depend very largely on a demonstration by lantern slides of various lesions of the esophagus and the viscera adjoining it. When I was asked to present this paper and take part in this symposium, I was a little loathe to do so, because it was as if I did not have any material. I went back over the material I had and was surprised to find that practically all of it consisted of X-ray examinations made years ago. I suspected that somebody was sending me a lot of esophagus cases at that time who evidently has ceased to send them. Almost all the plates that I am showing were taken between eight and ten years ago. I am therefore glad of the opportunity to present this, because it led me to realize that I am not making as thorough and complete an examination of the esophagus as I should in every case that comes in.

The methods of filling the esophagus are four. The first is the fluoroscopic observation of the bismuth meal as it passes into the esophagus, with the patient in the erect position; the second, the sausage-casing described by Stewart, tying one end and allowing the patient to swallow it, and then filling the sausage casing with barium; the third, one described by Hirsch as working a thick bismuth and barium solution with gum tragacanth, which, he maintains, sticks to the esophagus; and the fourth is what I employ, that of giving the patient such a thick paste that he cannot swallow it, and letting him keep it in his mouth as long as he can, and then making the examination rapidly. In that way I get a good contour of the entire esophagus.

Since I have been asked to present this paper I have done one other stunt, and that is letting the patient, after he has swallowed the barium meal for a stomach examination, retain three or four swallows in the glass, and then letting him take some of the thick paste and swallow that, and then take one or two swallows of the thin bismuth paste. Then, with the patient lying on his side when he takes this, you can get a good picture of the esophagus without the disagreeable part of swallowing the sausage casing.

The thing that was especially brought to my attention was the contour of the esophagus as influenced by anatomical structures bordering on it. These organs can be readily recognized in a moderately dilated esophagus. Consequently, in disease, alterations in size or pathological changes in many of these organs, I believe that we shall greatly increase our scope of mediastinal diagnosis by making an esophageal examination in all cases. Therefore I am distinctly happy that you have asked me to do this, because that is one of the things that I have gotten out of it. It is essential that we shall recognize these anatomical structures, in order not to mistake them for something in the walls of the gut. The X-ray examination is valuable in recognizing foreign bodies, spasms, dilatations and diverticula.

DISCUSSION.

(See page 154.)

(a) A METHOD OF X-RAY CONTROL IN THE RADIUM TREATMENT OF CANCER OF THE ESOPHAGUS.

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The beneficial action of radium on malignant tissue is due to its selective retrogressive action on the cancer cell when used in a dosage still not harmful to normal tissue. When attempting to utilize this principle in the practical therapeutic use of radium certain difficulties are encountered. First, what may be termed the coefficient of tolerance of normal as compared to pathological tissue is not as great as would be ideal and second that the devitalizing action of radium on malignant tissue rapidly decreases with its distance from such tissue. This decrease is approximately as the square of the distance. Not knowing the thickness of a malignant tumor it follows that it is desirable to use as strong a dose as will be tolerated by surrounding normal tissue incidentally exposed to its action. In cancer of the uterus, conditions are maximally favorable, as it is possible, on account of anatomical conditions to irradiate in the center of a mass, the cancer infiltrated uterus. Even if normal uterine tissue is injured, surrounding structures and reactions are such that protection is developed. The beneficial results of radium treatment in uterine carcinoma are due to the fact that under such conditions large dosage can be used. To cite another instance of effective radium therapeutics, where other conditions make possible other technical procedures. In superficial carcinoma, as of the lip, relatively large doses of radium can be utilized through centralization of application and protection of normal surrounding tissues by filters. The problem offered by carcinoma of the esophagus is quite different from that of other malignant conditions where favorable results have been obtained. We have to do with a tumor whose exact thickness is unknown and usually not uniform. Not only is there no surrounding tissue of a protective nature but instead the thin-walled esophagus in contact with vital structures whose devitalization may lead to ulceration and perforation. The situation of cancer of the esophagus renders exact centralization of application and protective procedures mechanically difficult.

There is as yet no accepted criterion as to the amount of irradiation that the normal esophagus will tolerate. While this would at first thought suggest a safely minimum dose it must be recalled that if benefit is to be derived the dose must be large enough to be definitely active throughout the entire tumor. This because smaller doses while favorably affecting immediately contiguous malignant tissue may reach more distant portions of the growth so weakened as to be stimulating rather than inhibiting. If there is to be a prospect of a cure of the disease it would seem dependent on a considerable dose being employed. The situation as regards cancer of the esophagus is not one that indicates temporizing. The disease saving possibly a rare surgical cure is fatal, its course attended by suffering and a distressing lessening of morale. If in any condition a treatment embodying an element of risk is justifiable it is so in this instance. It would seem that the maximum dose compatible with a fair degree of safety must be conceded as the primary requisite. The problem secondarily is one of precise application and protection. Regarding the first difficulty, it is hoped that this presentation may offer aid; of the second, it less definitely suggests possible help.

Requisites to the successful emplacement and continuous application of radium in cancer of the esophagus are, first, a knowledge of the location and physical peculiarities of the tumor and the resulting stricture especially as to its location, direction and the degree of stenosis. Second, a means of effective and nontraumatizing canalization of the cancerous stricture. Third, a mechanical means of maintaining the radium in direct application with the tumor. Fourth, a ready means of frequent observation as to the position of the radium during the period of treatment. Fifth, a careful selection as to dose, filtration and frequency of treatment guided by such experience as we have and the individual peculiarities of the case. Esophagoscopy has heretofore been utilized by some to accomplish certain of these ends. Esophagoscopy plus careful measurement can ascertain the location of the proximal portion of the tumor. Sounding by olive tip bougies or gastrostomy can serve to locate its probable distal boundary and give a general idea of the luminary inequalities of the stricture and determine its degree. Esophagoscopy can through recovery of tissue establish the diagnosis though not admirably according to present surgical teaching. Esophagoscopy might serve to map out the topography of the stricture in certain unusual instances in which it was not so great but that it could be canalized by the esophagoscope. It will also serve as a means of emplacing radium at least within the upper reaches of the cancerous stricture. Esophagoscopy can best determine tissue reaction after a radium treatment. The procedure has as its disadvantages: Those natural to such an instrumentation. That in most instances it cannot give a definite knowledge of tumor topography but only of the proximal end of the stricture. That it furnishes no means of ascertaining the permanences of location of the radium after it is initially placed without frequent instrumentation and then no clue as to its location and relation to the tumor when it is necessary to place it below the initial stricture, as is frequently the case.

The X-ray has advantages over esophagoscopy for certain of the purposes in question and may be successfully utilized to serve ends impossible by it; especially in that it furnishes means of frequent and convenient observation as to the location of the radium in any level of the stricture during the entire treatment. The foregoing is a very important matter as it has been found that the radium terminal almost invariably sooner or later slips into lower portions of the stricture or beyond it, less frequently above into the dilated esophagus. Again the use of fluoroscopy during the emplacement of the radium capsule is an effective means of aiding in the canalization of the cancerous stricture visualized by the patient's having just previously swallowed a small amount of bismuth in suspension.* It is frequently an advantage to bend the wire carrier slightly at its attachment to the radium capsule so that the terminal is deflected at a slight angle. By torsion on the oral end of the wire applicator and placing the patient at different angles the terminal may be guided directly into the stricture. In certain cases of marked cancerous stenosis the tip of the radium terminal may be made to engage within the proximal portion of the stricture but further progress is prevented by the degree of narrowing. In such instance under careful fluoroscopic control the tip may be left engaged in the stricture, the walls of the esophagus above being protected by the distention of the esophagus proximal to the stricture by a bismuth mixture. It will usually be found on later observations that owing to the continuous gentle pressure exerted by the spring of the wire applicator the radium gradually canalizes the lower levels of the stricture. In case the stricture is not visualized at any time it may be made apparent by the simple expedient of having the patient swallow a teaspoonful or two of bismuth media alongside the wire applicator.

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*Bismuth in small amounts gives a slightly heavier shadow than does barium.

It is difficult to understand how any approximation of the radium to the body of the tumor can be effected by other than some sort of visually controlled method; a matter that is fundamental and the importance of which is becoming more and more recognized. In many cases the strictured lumen is devious and opens from the proximally dilated esophagus to one side. It is remarkable what aid can be obtained by fluoroscopy. A threatened false passage is strikingly indicated. The cause of failure to canalize any portion of the stricture is shown to be usually due to impingement of the terminal against the wall of the stricture on account of an abrupt curve rather than to a local narrowing. Plates taken and developed between efforts at introduction and as a record of emplacement at any given time are helpful.

While less appealing, the X-ray diagnosis of cancer of the esophagus in expert hands taken in conjunction with the clinical history will probably attain as high a percentage of accuracies as can be realized by esophagoscopy. Occasionally the X-ray serves to visualize the actual shadow of the esophageal tumor silhouetted between those of the heart and spine. If a technic can be evolved that will do this with any constancy and especially in other than the diagonal poses, it will be of great advantage, as the actual extent and various thicknesses of the mass can be determined and the radium not only more effectively placed but a basis established for control of tissue protective unilateral filtration; this with the view of using larger dosage, when conditions make it desirable. Most hopeful in this regard is a technic developed along the same lines as in gall bladder work, namely strong fixation of the patient, the use of small diaphragms, extreme care as to the suspension of respiration and a careful technic of exposure adapted to the individual patient. Fast screens are best used to obtain speed in exposure necessitated on account of transmitted movement from the heart's impulse. The X-ray also serves as a means of determining the results of treatment in so far as the degree of stricture and to some extent of the change in the tumor is concerned. The X-ray control of irradiation of cancer of the esophagus has all the unique advantages and gives the peculiar satisfaction and surety that is characteristic of the use of the X-ray elsewhere. As an aside, the X-ray method of control may be used in other esophageal instrumentation such as the divulsion of strictures with opaque bougies and as we have used it for some years in the treatment of cardiospasm with a special bismuth containing lead mounted dilator. The dilatation of cardiospasm under roentgenoscopic control is a revelation.* In comparing esophagoscopy and the X-ray for the control of the treatment under discussion it is not implied that either alone should be relied upon if advantages possessed to either method are unique for certain purposes. Both should be used as indications arise.

The technique employed in the cases reported may be summarized. An initial X-ray study of the position and physical peculiarities of the tumor by both screen and plate using a simple mixture of bismuth subcarbonate in water and where the stricture was not great, bismuth suspended in artificial buttermilk as a means of visualization. If plates are taken an instant after the patient is told to swallow a mouthful of the barium containing substance previously held in the mouth while in pose the stricture and the distal esophagus will usually be visualized.** If the stricture is of high grade, the act of swallowing tends to force through it barium previously swallowed and retained above the stricture. When the stricture is less marked the fact that the contrast substance does not pass through the cardia as rapidly as usual, possibly through inhibition of the conductivity of the nervous impulses governing peristalsis is a help in visualizing the lower esophagus.

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*Fluoroscopic control of cardiospasm dilatation shows that it is difficult to cause the dilator to straddle the site of spasm and still more difficult to cause it to remain in place during the divulsion. It also shows that the best way to accomplish this is to introduce the dilator beyond the hiatus esophagi, to distend it very slightly and then pull it into the area of the stricture and while holding it in place with considerable traction inflate still more. The X-ray also strikingly shows that the standard dilator is much greater in diameter than is necessary. The spastic area is never dilated to the full diameter of the dilator at the time pain is elicited. Judging by those divulsions that have been done under fluoroscopic observation it is difficult to understand how dilatation of the spastic area could be accomplished to the full capacity of the dilator without danger of rupture of the esophagus. When full dilatation is accomplished the dilator is probably either above or below the seat of spasm. The moderate distension above referred to is entirely adequate to accomplish the most beneficial results.

**This time varies somewhat according to conditions in the individual case, but is usually about three seconds.

The patient is given a preliminary injection of morphine and atropin one-half hour before the radium treatment is begun, the dose obviously as indicated. It is impossible to overestimate the value of this procedure in quieting the patient and making the endurance of a six-hour application possible without undue suffering. Many patients sleep through most of the treatment. Occasionally in marked strictures a spoonful of olive oil one-half hour before treatment is helpful in relaxing secondary spasm. Preliminary bouginage is occasionally useful. The radium enclosed in a container composed of German silver one-half millimeter in thickness and further filtered with one-half millimeter of brass and a thickness of rubber is mounted as a terminal on a slightly springy drawn silver wire encased in a rubber tube. It is introduced after the manner of an ordinary esophageal sound. A wire applicator has been used by others. In certain cases where the abdominal esophagus was involved the radium with its filters was encased in the end of a rather small stomach tube, as it was sometimes found that the wire carrier could not readily and safely take the rather abrupt curve of this portion of the esophagus. The wire applicator or stomach tube bearer is anchored by means of a bridle bandage about the patient's head. The radium is left *in situ* for six hours at each initial treatment. Cases were treated on from one to seven occasions. The frequency and number of treatments and the length of other than the initial treatment was occasionally varied somewhat to meet individual indications; also as much as was thought advisable in an effort to determine the most effective procedure. Nearly all our work has been done with 50 milligrams of radium element. It is probable that the use of radium emanation not available to us would have advantages over the element on account of the more advantageous form of applicator that it would permit. Our greatest difficulty has been due to the thickness, length and rigidity of the radium containing receptacle with its filters which precluded satisfactory introduction in certain marked and irregular strictures.

The immediate results of the treatment were in most instances beneficial, sometimes strikingly so as to the relief of the dysphagia. No case treated failed of improvement in this regard.

The improvement in several was almost immediate, within 24 hours, possibly due in part to a bouginage action of the radium capsule. A gain in weight occurred in most cases. Curiously the results of treatment are not reflected by a reduction in the local esophageal deformity and stenosis commensurable with the functional improvement—possibly due to loss of local esophageal distensibility. In several cases there was a return in a degree of the dysphagia, usually relieved by another treatment. The re-established dysphagia in some instances seemed of the nature of intermittent spasm. The late return of the dysphagia may be due in certain cases to secondary contractures. It may be mentioned that radium burns result in dense scar. Possibly with added knowledge late careful dilatation by bougies might be beneficial; we have not attempted it.

The propriety of reporting and reviewing a small series of cases under observation but a short time may justly be questioned. On the other hand, we feel that this is advisable under the circumstances. If workers delay reporting cases until several years have passed and final conclusions as to the value of the method is reached, valuable time will indeed be lost. We can at least profit by each other's disasters which are usually prompt. The matter of dosage, too, can be helpfully influenced if cases showing immediate beneficial results are reported. Again, carcinoma of the esophagus is relatively rare. If we delay until any considerable series of radium treated cases is accumulated, the development of the method will be prolonged. A presentation of cases even with definite limitations will help develop an interest in the method and serve as an incentive to earlier diagnosis which is, after all, the prime requisite on which our hopes are built.

Our own tentative results may be summarized as follows: Fifteen cases treated. No case treated which might be considered a cure, though one patient is alive and in good shape eighteen months after the first treatment without evidence of metastasis.* The esophagoscope shows no appearance suggesting present carcinoma. Others have been under observation less than a year. No case treated could be considered early and most of them were frankly advanced. There was but one case in which there was evidence of metastases and in that instance there was involvement of the stomach as well as the abdominal esophagus. Five patients have died, one possibly of perforation resulting from the treatment. In six cases the dysphagia has been strikingly improved. In four additional cases the improvement in swallowing has been definite though intermittent. Seven cases were improved as to general condition. In certain of these generally improved cases the result was so striking as to indicate that the treatment is strongly advisable even where there is no possibility of a cure. There seems no question but that life is prolonged by the treatment. The patients are usually enabled to continue their work. Not the least of the benefits is the improvement in morale. The work, instead of being gruesome, is made gratifying by the pitiful appreciation of the patients.

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*At the time this article goes to print, January 24, 1921, the patient still shows no evidence of malignancy, two years and six months after the initial treatment. Another patient is alive without objective evidence of malignancy one year and six months after the initial treatment.

A mention of the results of others in the treatment of cancer of the esophagus by radium is fitting though these instances are few. In Exner's cases favorable results were considered indicated by decrease in the degree of stenosis as determined by bouginage. In Einhorn's series the majority of cases were palliatively improved and decrease in the local lesion was considered indicated in certain instances by increased distance of the stricture from the incisors. Janeway, Barringer and Failla may be quoted as follows: "The series of cases reported by no means justifies a gloomy outlook. Cases too late for a cure by radium can receive palliative relief. But one case was sufficiently benefited to encourage belief in complete retrogression. (This case had been under observation a little over a year.) Five cases were definitely improved. One case which improved had an advanced lesion at the time treatment was begun. Fifteen cases were not improved; the authors felt on account of the advanced state of the lesion and to too frequent treatments. A. E. Pinch reports a case of epithelioma of the esophagus treated by radium. The patient lived one year, dying from metastases. As far as the local condition was concerned the treatment was eminently satisfactory, 'infinitely better than could have been obtained by gastrostomy.' Chevalier Jackson mentions having treated cases of cancer of the esophagus by radium and states that he has seen marked favorable effects in inoperable cases, but so far no cures. He prefers to wait several years before passing judgment. He speaks of the helpfulness of surgery in the condition and of having seen no case amenable to operative treatment in which he would consider the use of radium justified. He mentions Mr. Walter G. Howarth as obtaining excellent results by the method. C. W. Hanford obtained material benefit in one of the two cases treated by him.

The question of the treatment of cancer of the esophagus by radium versus surgical treatment should be mentioned. If the lesion is definitely early favorably situated and the rare surgical experience available, surgery should be the method of choice. Actually such a combination will rarely occur. Possibly as the importance of early diagnosis becomes more appreciated and the surgery of the lesion is developed operative treatment will be more frequent.

As to palliative treatment the indications are that radium therapy is the best that has yet been suggested; at least patients may live to die of less agonizing consequences than result from esophageal stenosis. From a consideration of all factors the possibility of the ultimate cure of certain cases of cancer of the esophagus by radium must be held a definite possibility.

In conclusion: The method of treating cancer of the esophagus by radium is hopeful. The most encouraging feature in the small series of cases treated being the relief of the dysphagia. The X-ray gives valuable help through establishing a knowledge of the peculiarities of the individual lesion, as an aid in the emplacement of the radium and in the control of its localization. Its use suggests other possibilities as to its employment not only in the treatment of cancer of the esophagus by radium but in other esophageal instrumentations.

DISCUSSION.

(See page 154.)

(b) A METHOD OF RECORDING AND STUDYING INDIVIDUAL VISCERAL TOPOGRAPHY WITH THE PATIENT RECUMBENT.

To my knowledge there has never been an effort made to apply certain methods of graphic record now available, such as photography and orthodiography to the study of topographical anatomy. Such illustrations as we have, attempting to depict visceral topography on an X-ray basis, are unpretentious schematic drawings of the rubber stamp variety made with the patient presumably in the erect posture. The old order of the depiction of abdominal and thoracic topography from post-mortem findings as illustrated in all text-books still obtains. It would seem that individual, and as a resultant, type topographical studies of conditions as they obtain in the living with the subject recumbent as made possible by the modern means mentioned, are very desirable.

After various efforts a means of so graphically recording the facts of general physical and topographical anatomy with a fair degree of accuracy and by a simple method seems to have been effected. It should be emphasized that such a means of record should be a comparatively simple process. If an elaborate and complicated method is evolved it may be effectively used by the originator, but will have no field of usefulness outside of his efforts. A method of the kind under discussion should require no special training in any line. It can, of course, be carried out more effectively if one has a certain amount of facility with pencil and camera; however, for practical purposes, tracings need not be elaborate or pretentious and nothing without the easy range of those accustomed to X-ray work is required.

The method to be described consists essentially of the photographing of the recumbent patient by means of a camera attached to the ceiling and with a sheet of glass on which are orthodiagraphic tracings lying on the patient's abdomen and so interposed between his body and the camera. The visceral outlines are on the sheet of glass, but glass being transparent, if arranged in certain fashion is not apparent; the lines on the glass appearing as though marked on the body.

While the foregoing statements indicate a simple procedure, a good many technical difficulties were encountered in its practical development. Chief among these was the difficulty of obtaining a source of artificial illumination that would not throw a reflection on the superimposed glass on which were the tracings. A satisfactory arrangement was finally effected by utilizing the principle of the multiple Bartlett lighting system used in operating rooms of having a circlet of electric lights overhead large enough so that none of these lights was directly over the glass plate. An important matter also is to see that the tracing glass is level at the time the exposure is made. Another difficulty was that the lines on the interposed tracing glass threw shadows on the surface of the body. This difficulty was obviated by having the electric lights furnished with reflectors whose parabolic curves were such that all focused in the plane of the tracing glass so that there were no shadows. It might be mentioned that the overhead camera was arranged on a vertical sliding mechanism so that it could be lowered for inserting plates. The camera was locked in focus so that when elevated to a certain point it always gave a sharp focus without other than the initial adjustment. This latter, together with the fact that an artificial lighting system was used, reduced the technic to a very simple matter. The camera was lensed and otherwise so arranged that all figures were reduced to exactly one-third natural size—this for purposes of comparison and measurement. A good deal of difficulty was experienced in obtaining a proper medium for the tracings that would effectively show on the body. The best results were obtained with the old Faber's china pencil No. 2251 for white people and an American made white china pencil in the case of negroes and others with dark skins. This pencil was Blaisdell No. 164.

For purposes of record prints may be made from the photographic negatives obtained as above described. These prints may be lettered, drawn upon. Written or better typed legends may be added as seems best. The finished prints make admirable permanent records and for brevity and conciseness of information will hardly be equaled by any written description.

It has been mentioned that such studies should be made with the patient recumbent because recumbency is the posture in which of necessity the physical examination of the abdomen must be made and consequently the facts of visceral topography as determined by the X-ray must be correlated.

In addition to a more exact knowledge of normal visceral topographical anatomy a method for summarizing, studying and graphically recording pathological topographical changes seems needed for diagnositc purposes. Such a method should show changed topography as, for instance, resulting from pressure, contractures, fixations by tumors, exudates, ascites and the like. It should be a method by which the findings of physical examination, of inspection palpation and determination of points of tenderness, may be correlated with information obtained through the use of the X-ray corrected by orthodiography. Such a method of photoorthodiography should be of helpfulness to the surgeon by summarizing information obtained through the various means mentioned and making it immediately available at the table in the individual case. It should show the relationship of the viscera to surface landmarks and hence indicate the most advantageous site of incision. It should show the structures sequentially to be encountered and especially indicate the topography of adjacent structures with reference to the incision. It should indicate unusual fixations and abnormal positions of parts. Especially it should be a means of information as to the probable feasibility of radical surgical procedures, as resections and anastomoses and their most favorable locations.

DISCUSSION.

Dr. J. C. Hemmeter, Baltimore, Md.: Dr. Mills has brought out some admirable points. Some years ago, I reported some measurements of the human body in different locations in the gastro-intestinal tract. I should like to ask Dr. Mills to continue his observations in connection with a paper that I brought before the society some years ago, on Anthropometric Measurements. He has done some work on that same kind of measurements in connection with the osseous system. Such anthropometric measurements should be based on the canon of human proportions laid down by artist-anatomist Leonardo da Vinci and Albrecht Dürer.

THE SURGERY OF DISEASES OF THE ESOPHAGUS.

By Dr. Willy Meyer,

NEW YORK.

The surgery of the esophagus is a very large chapter. I do hope that it will meet with the approval of the president and the members of our Association if I venture to pick out one chapter only and say a few words on the surgical treatment of cancer of the esophagus.

Let me first recall to your mind that cancer develops with particular frequency at the site of the so-called physiological strictures—behind the cricoid cartilage; behind the bifurcation of the trachea, and at the cardia. Unfortunately, these places are the hardest at which the condition can be attacked surgically. The very best location of the cancer for the surgeon is between the arotic arch and the cardia. But here it is rather rarely found.

The affection is more frequently found in the male than in the female; however, in the last few years I have seen it quite often in the female. It occurs particularly in two types—the squamous-celled epithelioma and the infiltrating cancer made up of cylindrical epithelial cells. The latter forms rapidly large tumors, covering from two to four inches of the tube.

Permit me to recall to your mind, further, that cancer of the esophagus is considered by all pathologists to be the most benign of all carcinomas, affecting the lymph nodes late, and that in case of the clinical signs of stricture of the esophagus, including all types, more than 90 per cent. belong to the class of malignant strictures. This, to my mind, is of great importance to remember. Usually these patients present themselves first to the family physician or to the specialist. After the doctor has found out that there has been no swallowing of an acid or caustic lye, that there has been in the family or previously in the patient himself no tuberculosis, and no suspicion of syphilis, let him primarily think of carcinoma; let him know that after this suspicion has arisen in his mind, these cases should be considered as much important surgical cases as those of acute appendicitis.

Where should the patient be sent to? To the hospital, and only to such hospital where every necessary examination can be carried out. I am speaking of patients without means—ward patients—because other patients can have private examinations. But if a doctor has such a patient, let him take care that he is rushed to the hospital, losing not more than a few days for all the newer examinations required.

What are the newer examinations that can help us to make an early diagnosis of cancer of the esophagus? We know that in the advanced cases, such as we have considered this afternoon, the diagnosis can be made by means of the X-ray and the opaque meal. The last few years have seen the construction of aids in the early diagnosis of this condition, particularly of sounds. They work in a retrograde direction, inasmuch as they pass the stricture when pushed forward. On being gradually and slowly withdrawn, the instrument will make the examiner note resistance at a certain place. The X-rays cannot show and make a definite diagnosis of an early stricture if done in the usual way. However, the experiments made by Dr. A. S. Crump, of New York, with hog skin and bismuth ingestion promise to give us great assistance in the early diagnosis. The esophagoscope is also of great assistance. By its use experts may discover a suspicious place in which the condition is not normal.

I have, of course, *not* in mind patients in whom the carcinomatous stricture has already completely surrounded the esophagus. Cancer of the esophagus will develop like cancer of the colon, in a crescent, and only gradually surround the entire organ.

If all these things have failed to refine the definite diagnosis, we have exploration. Surgeons believe that the exploratory thoracotomy is just as safe as exploratory laparotomy. In the last few years I have had a few far-advanced cases in whom I could not do more than make an exploratory incision for thorough exploration, followed by airtight drainage. Every one made a most gratifying recovery. As long as we have asepsis at our command, as long as we adhere in our work to the fundamental principles of thoracic surgery, we may consider exploratory thoracotomy as safe as exploratory laparotomy.

All surgeons who have shown interest in the operative treatment of cancer of the esophagus agree that up to this time we had only far-advanced cases referred to us, and everyone has attacked these cases surgically with enthusiasm, doing much more than should have been done. We would not resect a cancer of the pylorus that had invaded the pancreas or the liver; we would do a gastro-enterostomy. We have done too much surgery in cancer of the esophagus. We have gone too far, with results that had to be negative; but in doing so we have learned how to do it. We have learned the way of operating. We also know how to treat the patient after operating; and we have tried to avoid that threatening condition after every operation—pneumonia.

We do not walk in the dark any longer when we attack the esophagus. We expose the tumor; if it is operable, we go ahead. We invert the distal end and transpose the proximal by means of the so-called extrapleural superior esophagoplasty. We then connect the esophagus with the gastric fistula, and the patient can again swallow food.

One word more. Whoever has treated to the end a patient with inoperable carcinoma of the esophagus, and has heard day after day the question, "Doctor, when shall I again be able to swallow?" will likely be ready to do a palliative operation. Divide the esophagus above the tumor, which latter is left in place undisturbed; invert the distal stump, bring the proximal out at the neck, and unite this to the gastric fistula.

I could show some slides of a case in which I did this in a patient weighing 89 pounds. I did it in order to give her the ability to swallow. The happiness she exhibited when we joined the two ends by a tube, so that she was able to swallow once more, is one of the most pleasant memories of my life. This, then, might be done in inoperable cases. But if we get these patients in time, surgeons will, no doubt, soon be able to show a series of cases of operative cures similar to the one reported by Torek of the Lenox Hill Hospital of New York. This patient came directly to the surgeon; she had not passed from dispensary to dispensary, from clinic to clinic, and from hospital to hospital for many months, eventually having been dilated, and consequently made inoperable. Torek's case was really operable; and that patient is still alive today, completely cured, having been operated on in 1913. If surgeons would get such cases oftener, similar favorable results might soon be shown.

The principal point is—and I would like to emphasize this once more—that today we know how to do the operation and how to conduct the after treatment.

One word yet regarding radium. I do not believe that radium will *cure* many patients afflicted with esophageal carcinoma; it may alleviate the symptoms quite often. But it should be borne in mind that radium application within the esophagus is dangerous on account of a burn. Sloughing may cause a fatal posterior mediastinitis. It is my belief that internist and surgeon should compromise. As soon as the advance made in this chapter enables the internist to make a really *"early"* diagnosis of cancer of the esophagus, let him give the surgeon the chance of an *early* operation. I feel sure that a series of successful operations of resection of the esophagus *with lasting results* would soon be reported.

DISCUSSION OF PAPERS OF DRS. FREEMAN, SMITHIES, COLE, MILLS AND MEYER—SYMPOSIUM ON THE DISEASES OF THE ESOPHAGUS.

Dr. F. H. Baetjer, Baltimore, Md.: I can only talk on this subject from one standpoint, and that is the results obtained from X-ray examinations. I was much interested in Dr. Freeman's discussion on the use of the esophagoscope and Dr. Mills' criticism that with such an instrument only one end of the lesion could be seen. I have seen two cases in which the examination by the esophagoscope was absolutely negative, and yet the barium bismuth examination showed small ulcerative lesions at the cardiac end of the esophagus. The only way that the negative result from the esophagoscopic examination could be accounted for was that the end of the instrument had probably pushed a fold of mucous membrane over the ulcerative lesion and thus it was not seen. Then, too, I have seen several cases where there was absolutely no history of any difficulty in swallowing and where one would not be led to use the esophagoscope and yet the X-ray examination has shown lesions at the cardiac end of the esophagus.

Dr. Cole covered the point so thoroughly in regard to the various methods of examination that this subject need not be mentioned, except to say that I employ one additional method. He spoke of these as a memorial of the happy days before January, 1918 (?). I get the patient to take a mouthful of bismuth, and immediately a second. By doing this, you will find that instead of the ordinary distance between the two swallows being maintained, the second will catch up with the first swallow, showing that there has been an arrest at a certain point. This does not obtain with ordinary physiological narrowing of the esophagus.

With regard to X-ray examination, it is questionable whether we can make an absolute diagnosis from the X-ray alone; but that is only one of the many methods of examination.

Dr. Smithies has pointed out the difference between the cardiospasm and the carcinoma. That can be explained on mechanical grounds. Just as Dr. Meyer said, when a carcinoma starts, it does not at once surround the esophagus. It increases in size at the spot where it originally starts, and that throws the opening of the esophagus to one side.

Another point is that which Dr. Meyer brought up, that of metastases to the lung. I saw several cases of this, one of which is worthy of note. Examination showed irregular edges of the growth in the esophagus, and also a very extensive tuberculosis of the lung. There was no question as to tuberculosis of the apices, but both bases were also involved, showing large, nodular masses. That patient died, and the lungs were removed as a whole and X-rayed; and then we cut down on the nodules and X-rayed them again. We identified each nodule, and then made microscopic examinations. Some were found to be malignant, and some tuberculous.

You do not expect to see carcinoma in childhood. That is the age of foreign bodies and traumatic strictures. You do not expect to see cardiospasms. In one child of seven, there was what seemed to be a cardiospasm. Then take the period of early middle age. It is in this period that I have seen most of the cardiospasms and evanescent spasms. In old age we have most of the malignant growths, and also of luetic lesions. I have seen some cases in old people that I thought to be carcinomatous clear up under antiluetic treatment. I was not able to differentiate between the two conditions.

Dr. Max Einhorn, New York City: With regard to Dr. Smithies' paper on cardiospasm, I must say that in most of the cases in which there is dilatation of the esophagus present, stretching of the cardia can be done with the blunt instrument by the direct route to the stomach. We can push the instrument to the stomach. I do not think that the spasm really exists in all cases. There may have been a spasm; but when the esophagus is dilated, the cardiospasm is found to be but a nervous defect. The cardia fails to respond to the opening reflex, and, in consequence, everything accumulates in the esophagus. While the cardia itself is not tightly closed, the fluid cannot push itself through. In pushing in a bougie, you meet with no resistance at all, and can go directly into the stomach. Some of us do, and meet with resistance there, and pass through that, too.

The frequency af late cardiospasm is much greater than cases in which the instrument fails to enter the stomach and engages in the wall of the esophagus, or meets the cardia and cannot pass it. I should think that nine out of ten are cardiospasm, and that only one-quarter of these are cases in which you cannot go in with instruments.

The cases are easily managed, and, by stretching the cardia, they get cured. We make the opening large, so that the walls do not lie on each other. Malignant strictures should never be stretched, because the stretching irritates the growth. I do not do it now; but at the beginning of my practice I stretched a few cases and noticed that they developed fever and went down very rapidly. I suggest that in malignancy you should not stretch the cardia. It is different, if the malignant disease is not at the cardia, but, perhaps farther on in the stomach. In that case the stretching would not do so much harm; but these cases can be differentiated because they have a different history.

Regarding the treatment by radium, which Dr. Mills has spoken of, I am much gratified to find that he can put the radium down upon the stricture. I had thought that it was applied at the beginning of the stricture. I have found that in half the cases we achieve good results—temporary results, at least. The patients can eat, and they live a little longer. But by the method Dr. Mills has devised, we can go a little farther; and where there are extensive strictures we can go in. His method of application seems a little more efficient than the old one, and I should think that better results would be accomplished by it. I am not sure that we could not succeed in curing patients with radium. It may be that we shall succeed. The cancer of the esophagus does not show a tendency to infect the lymphatic glands, but it has a tendency to localize itself; and if we should start the treatment very early, we might be successful. I do not see why not. Tumor of the eyelid has been cured by radium applications. I have such a case myself, though I am not an eye specialist. The patient had a big nose and eyelid epithelioma. I sent him to Dr. Denig, the eye specialist, who said that he could not do anything, and as the skin specialist could not do anything either, we decided to try radium treatment. He had a few treatments in the hospital, and I continued the treatments for a while. The patient was cured, and has remained so. I believe that Dr. Denig showed that case before the Clinical Society of the Lenox Hill Hospital, New York.

DR. SIPPY (*closing*): Dr. Mills spoke of the difficulty of making the bag stay where it should. We use this type of bag, pass it over the tube, and can always put it where we wish to put it. The dilating force is here. We found, in working on dogs, that the esophagus could be ruptured readily. We never go over 250 mm., as measured by the ordinary blood-pressure apparatus. It is not a firm contraction that we have to overcome. The muscle is very thin. If you pump it to two hundred millimeters, it will make firm pressure, but will not rupture the esophagus.

The patients rarely die. In the picture that I sent around, the patient did die from starvation. She had been operated on, and came to me nearly dead. We made the diagnosis, and the next day she became flgihtly from inanition; and though we made a gastroenterostomy to feed her and fed her for four days, she went on with the intoxication of inanition and died.

You see there a gastroenterostomy and gastrostomy operation, and the original cardiospasm that was not diagnosed.

We measure the pressure, and do not rupture the esophagus. The dog's esophagus will hold 300 millimeters of pressure. We feel that 150 or 200 millimeters is all that we dare use, and that is sufficient.

Dr. S. J. Meltzer, New York City (closing): The word "cardiospasm" is a misnomer. It was introduced by Mikulicz in 1881, and he recognized later that he was mistaken. He thought at first that the cardia was open; but he introduced a cardioscope and found that it was closed, and thought that the condition was a contraction or a stricture. That is a wrong name. Last year I presented a paper before the Association of American Physicians on the subject. Thirty-three years ago, however, although I did not want to coin a name for it, I described the disease, and last year I called it functional permeability of the cardia.

There is in the cardia a moderate loss of the tonus of the cavity. The food usually remains there a few seconds, until the contraction of the lower part of the esophagus, being stronger than the tonus of the cardia, overcomes it and drives the food into it. It may happen that the tonus has a degree of spasm, or it may happen that the lower part of the esophagus does not have the ability to contract, and so cannot overcome the stasis. There is no spasm at all. On the contrary, it is less than the normal contracting power. It is an anatomical cause, and was described years ago. What we are now terming cardiospasm is really not a spasm. Some cases may be spasms, but they usually are a disturbance in the functional conditions—the ability of the lower part of the esophagus to contract, or the tonus of the cardia. Either the tonus is increased or the tonus is diminished.

Dr. Smithies (closing): In my paper you will find the description of all, or most, of the facts brought out by Dr. Meltzer. I shall refer to these in the future as "Meltzer's syndrome."

Dr. Mills (closing): While the hour is late, I feel that I must inflict one or two things on you. Dr. Meyer did not touch on a point that is, of course, a very serious one; and that is, the use of radium *versus* surgery, in cancer of the esophagus. In my typed paper, I make this statement: "If the lesion is detected early, surgery should be the method of choice; provided that the unusual surgical skill and experience requisite is available." Such skill and conditions are rare, of course. Now consequently the present is the time to ascertain, in cases where such service cannot be commanded, whether the condition can be safely treated and helpfully influenced or even cured by radium. I believe that it can, if the case is obtained early. There is no lack of precedent concerning the curability of superficial epithelioma by radium. Lesions of the esophagus resemble other growths amenable to radium therapy. Another favorable factor is the late formation of metastasis in carcinoma of the esophagus. In most cases the specialized skill necessary is not available; not everyone can go to New York, and so far as I know the technique has not been developed elsewhere.

I cannot refrain from mentioning again the method of controlling the use of radium with the X-ray; it is of great help. I have treated only 15 cases of cancer of the esophagus, which is indeed not so common, but in no case was injury done. It is difficult to understand how a cardiospasm could be ruptured, if the procedure were controlled by the X-ray. The whole thing hinges on the point of early diagnosis as in cancer generally. It is sad that the patients are seen by physicians who give medicine in the early operable stages of cancer; bromides in cancer of the esophagus, pepsin when of the stomach and pile ointment when it involves the rectum. What is needed is an urgent propaganda along the lines of that developed in pulmonary tuberculosis.

DR. MEYER (closing): I was glad to hear Dr. Mills make the remarks he did about surgery and radium in the treatment of cancer of the esophagus. To estimate the amount of radium required is most difficult. In the hands of experts the method may be successful, but at the spring meeting of the New York Society for Thoracic Surgery, Dr. Janeway spoke of 35 cases that he had treated with radium, none of which had really been cured. Since then I have heard of one or two in which he thinks that he has had a good result, the same as Dr. Mills.

On the other hand, if we get these cases early and knowing today how to operate and how to conduct the treatment, I think that a radical operation can be done with much greater hope of finding a permanent cure than with the use of radium.

Dr. Meltzer rightly asked how surgery could ever make progress if early cases were not referred for operation. Give surgery a chance. To my mind early cases of cancer of the esophagus should be operated on, and advanced cases treated by means of radium.

THE USE OF THE ROENTGEN RAY IN THE STUDY OF THE PATHOLOGICAL GALL-BLADDER.

By Arial W. George, M. D.,

BOSTON, MASS.

It was with a great deal of pleasure that I accepted the invitation of your vice president, Dr. White, to discuss with you the problems connected with the study of the biliary tract by the use of the X-ray. Frankly, I think this Association has conferred a very definite honor on our specialty. Several years ago I had the pleasure of discussing with you the problems connected with the diagnosis of ulcer of the duodenum. Tonight we are to discuss the study of the pathological gall-bladder. As you all appreciate, this has been a very difficult problem from the very beginning.

We X-ray men had a heritage handed down to us to the effect that gall stones could not be demonstrated on the photographic plate except in rare instances. But those of us who studied the duodenum as outlined in the early days by our good friend, Dr. Cole, began to see occasionally in the more and more perfect plates obtained of this region, shadows that were without question due to gall stones. With more careful study of this area as time went on, shadows were noted which were not definite and which did not appear to be accidental, but which were entities in themselves. We came to speak of these shadows as "suspicious shadows," and we would so report them to our consultants, in the hope that if the case were operated upon for some other definite lesion, at least some inspection would be made of the gall-bladder region. We were much interested to find in a very large percentage of cases that we were right, and that these same "suspicious shadows" were beginning to have diagnostic value. It is now history, how we tried with all the enthusiasm possible to present the work in the best way we knew how before our own society; our efforts were met sometimes with ridicule, most of the time with doubt and skepticism. Our published work was subjected to definite criticism. The continental school considered the work almost that of charlatanism. This criticism was most valuable. It had the effect of making us persist in our effort to demonstrate gall stones. We found that in a number of these cases in which "suspicious shadows" were found the shape of the gall-bladder was outlined; and the time came when we reported gall stones on these suggestive or suspicious shadows, and at operation the gall-bladder was found to contain not stones but pathological bile and thickened walls.

The question then immediately arose, Is it possible to demonstrate the normal gall-bladder? We all appreciate that the technique for the study of the gastrointestinal tract has progressed very remarkably since the work of Cole started. The results of the operative findings were so constantly good and beyond the averages that were accepted up to that time, that we felt, as we do now as strongly as ever, that with the plate method (not with injection of the abdomen with gas, but with the classical means of studying the gastrointestinal tract with the barium meal) when a shadow is visualized, of the size, shape and position of the gall-bladder, and this shadow is not accidental, not a lobe of the liver or the antrum of the stomach full of food, or one of the other organs accidentally of the shape of the gall-bladder, that we are dealing with a pathological gall-bladder. The question of stones became of secondary importance unless they showed clearly. This immediately opened the very interesting study as to why the gall-bladder should show only when the bile was pathological. We all will admit that bile has a normal color with slight variations; that when it becomes dark in color this represents pathology somewhere within the biliary tract. We found that when the specific gravity of this bile is taken it varies all the way from the normal up to 1.080. As a rule, the darker the bile becomes the denser it becomes, so there is a type of bile we call the dark tarry bile. You, gentlemen, will appreciate better than I the clinical symptom complex that arises in a case of this sort. It seems a very difficult problem to me—possibly not so much so to you—to diagnose clinically the degrees of pathology that the gall-bladder may contain without stones. This type of case, I know from experience, gives rise to many indirect gastric and intestinal symptoms without direct reference to the gall-bladder. Many cases are X-rayed that are simply cases of chronic cholecystitis, and the report goes to the consultant: "Negative gastrointestinal examination." And yet the consultant knows from his study that the case is suffering from some chronic gastrointestinal disturbance.

I have had the feeling for a long time that the gall-bladder is a source of systemic infection, as the teeth, tonsils, or any of the other organs that have been featured in medical literature recently. Up to date, the only means of determining these factors is the use of the X-ray and this is not 100 per cent. accurate.

From what has already been said, it seems that our first definite proposition is that the normal gall-bladder cannot be demonstrated routinely. If this is true—and it is not admitted by all X-ray men—then when we visualize a shadow which is of the gall-bladder or due to the gall-bladder, this gall-bladder to us is pathological, whether it contains stones or not.

Another point immediately arises in the study of the pathological gall-bladder, in such a case as above mentioned, when the surgeon after inspection and palpation at the time of operation declares the gall-bladder to be normal. Aside from a surgeon who does a good deal of surgery of this region, probably I see as many operative gall-bladders as the average man, and it has been my experience that it is impossible to be sure that the gall-bladder is normal merely from inspection and palpation. So this immediately opens the problem of surgery of this gall-bladder. We are of the opinion that when the time comes that a given case is advised to have surgery, not only from X-ray but clinical evidence, the surgeon falls short of his duty in a sense, regardless of his opinion, if he does not open the gall-bladder. I should like to go so far as to say that everything pointing to positive evidence, as well as negative evidence in other parts of the tract, this gall-bladder should be removed. Possibly I take a chance in saying so before this society when I might not if it were a body of surgical men; for when this matter has been brought before the different surgical societies I have been privileged to discuss it with, it has been the unanimous opinion after a certain amount of discussion, that this procedure is the logical one under the circumstances. We have found so far as our correct diagnoses go, that they were very much higher when the gall-bladder has been removed and the absolute proof made, than when the surgeon merely inspected and palpated the gall-bladder and backed out of operation.

Our cases today are studied and reported individually. We have no interest in statistics; we have no interest in studying these cases from a statistical point of view whatsoever. We all strive for 100 per cent. I do not believe that we shall ever reach that stage of perfection, any sooner in this study than we shall in any study pertaining to medicine; but there are so many diagnostic points of value in the study of the individual case that I want to take your time just to enumerate the important ones.

We have for convenience divided the diagnostic indications of the pathological gall-bladder into, first, direct visualization of the gall-bladder; second, indirect demonstration of the effects of a pathological gall-bladder; third, secondary manifestations of the pathological gall-bladder of the stomach, first and second portions of the duodenum, and the small and large bowel. It is only when we study the individual case from this point of view that we are able to consider the negative aspect of this study, and it is only then that the negative aspect becomes of value. Of this I shall speak later.

The direct visualization of the pathological gall-bladder, as has already been said, depends upon picking out a shadow which is that of the gall-bladder; one that is not of the stomach, or of the duodenum, or of the kidney, or of the liver; but one which through a series of films or plates remains relatively consistent. No one can tell beforehand whether only one plate will be necessary or whether ten will be required. One great requirement in the individual operator is perseverance, and he must persist in making a series of plates that are technically acceptable for this particular part of the tract. The plates must be made rapidly, must not be over-penetrated or under-penetrated, and must be taken in the same position as far as possible. It is not possible even with as much experience as we have had and with the development in the technique made by our operators, to obtain technically perfect plates of this region in any given case. The size of the individual does not bother us. Good plates are dependent more upon the ability of the patient to hold his or her breath than upon any other factor.

The indirect evidence to us is as important as the direct visualization of the gall-bladder. The commonest of all is fixation of the stomach to the subhepatic region; second, pressure defects upon the duodenum (first portion). Pressure against the antrum of the stomach when it is full of barium; deformities of the second portion of the duodenum in which it has been displaced or fixed, or picked up as we say; the displacement of the colon which occurs sometimes in hydrops of the gall-bladder. All these are important points in the final diagnosis.

One of the secondary manifestations, if we may be permitted to use that term, is finding the jejunum transferred from the left side to the right with the bowel empty, showing an accumulation of gas in various coils of the jejunum, or to find the jejunum when full of barium well up on the right side in the right quadrant, when normally it belongs in the left.

One other sign important to use, and upon which we have come to lay a serious amount of stress, is the filling of the ampulla of Vater with barium. I am not prepared to give figures, but I should say that in a series of 25 such cases where surgery has been performed, there was found without exception either a pathological gall-bladder or pathology in the pancreas, so this sign is a most important indication even when the direct and indirect signs are lacking.

The next important secondary manifestations are changes in the position of fixed points and deformities of the hepatic flexure and proximal transverse colon. And again, our *only* diagnostic sign in a given case may be this change.

The question will be asked unless I anticipate it (as I have found by experience) that the X-ray has no negative value in the study of pathology in the biliary tract. I think if we stop just for a moment and consider an individual case, taking up the points above mentioned: the direct visualization, the indirect signs and secondary manifestations, we shall find that all these signs may fail in a given case, but that our percentage of error on the negative side grows less and less as we consider these points seriously. In a large series of cases studied collectively, it is very rarely that a case goes through our hands with all signs lacking, and surgery proves some definite pathology. This has occurred, but it has occurred so rarely that I feel warranted in emphasizing this. Though I don't for a moment lay the stress on the negative side of this question that I do on the positive, still I feel that we have in the use of the X-ray a valuable means of demonstrating pathology, and a very helpful means of giving a negative opinion.

Before showing the lantern slides to illustrate the various points brought up in this discussion, I want to speak to you just for a moment on a few points pertaining to the problem of the study of the gastro-intestinal tract. You gentlemen are a group of trained men, trained in this specialty by study covering long periods of time and an extensive experience, and I am sure you study your cases very completely before you ask for X-rays. But the X-ray man, of like experience in his own specialty, and trained perhaps as thoroughly in his own specialty, is seeing your patient only once. He has to accomplish in his examination what it may have taken you months of study in the individual case and considerable laboratory work to determine—the possibility of pathology. Again, I feel sure that our only endeavor as X-ray men in the study of the individual case is to show you, if possible, by demonstration on the photographic film, all the variations from the normal in the gastro-intestinal tract, and to indicate to the best of our ability what these variations mean and whether they indicate pathology or not. Instead of being supercritical of the X-ray man's effort, try to appreciate that he is helping you secondarily, and primarily trying to help towards the diagnosis which eventually will mean the relief and cure of the patient. It is my hope that your efforts will be spent through suggestions and the results of your medical and surgical findings, in helping the X-ray man to be more exact, to take this problem more seriously, to actually do more work, and to develop two definite senses: first, perseverance, and second, perhaps using the term loosely, a little imagination. You must be always watching and criticizing your X-ray man who does not carry out the highest degree of technique but help him who is willing to strive to produce technically the best work.

I cannot close these remarks without adding what I have already said many times, that for all practical purposes the use of the fluoroscope is valueless in this study. If we depended upon the fluoroscope alone in the study of the gastrointestinal tract, as seems to be the inclination of a large group of men today, I am afraid it would be found to be of about the same value relatively as the stomach tube has proved.

There are a few men today who are carrying out the study of the pathological gall-bladder with the use of the X-ray to such an extent that if you care to consider statistics, I believe you will find they have an 85 per cent. of correct diagnoses in the study of this difficult problem. Your endeavor and my endeavor is to one end: the correct diagnosis and the cure of the patient, whether it be medically or whether it be surgically; for after all is said and done, even though it be humanly possible for us to be 100 per cent. correct in the study of the pathological gall-bladder, you gentlemen must consider whether the symptoms from which the patient is suffering are due to the gall-bladder or to some other form of pathology in the gastrointestinal tract, and it has always been my opinion that we, unbiased as we must be in the study of these cases without prolonged contact and study, and you who have more of the general facts at hand about the individual case can arrive at a better, clearer conception if both the X-ray and clinical evidence are taken into consideration and each given its due value.

DISCUSSION.

Dr. Lewis Gregory Cole, New York City: In my discussion of Dr. Robert's paper this morning I did not intend, as Dr. George has remarked, to "spill the beans." I fear that some of you may have the idea that I am at variance with Dr. George, but I am not, in any material way. I have listened carefully to everything he said tonight, although I have heard him say it before and have discussed it with him. I do not think that he said anything that I can in the least take exception to. The only thing that has disturbed me is his reference to shadows that are less distinct than any of the pathological gall-bladders that he demonstrated here tonight, or that Dr. Roberts demonstrated this afternoon. If I were to have the data that he submitted and that Dr. Roberts submitted, and should say, "We will agree to a diagnosis of a pathological gall-bladder in this case," I do not think that there would be any doubt at all but that the diagnosis would be correct. If I were seeing the plates that he has so graphically demonstrated here tonight—I am referring particularly to the pathological gall-bladder—I should not differ with him one iota, except in regard to gall-bladder shadows which show less distinctly than those he demonstrated.

If, in an effort to do good work and study the detail which we get in these plates, we see these shadows which simulate those which Dr. George has shown and are not so distinct as his, the question arises in my mind and the thing which disturbs me is, What shall we say about them? That is the only point about which there is any question of difference.

Since the morning session we have gone over ten or twelve plates of my series and, with one or two exceptions, both Dr. George and Dr. Roberts said that they would not consider this as evidence of a pathological gall-bladder, which I am quite convinced is the proper interpretation.

Dr. Roberts, under whom I had the pleasure of serving at Base

Hospital No. 1, where we discussed these questions—that is, gall-bladder diagnosis and the diagnosis of pulmonary tuberculosis by X-ray—made the statement that, in true pulmonary tuberculosis, there is never any doubt about the diagnosis from X-ray plates, and that when one makes an effort to over-study the plates one is on dangerously thin ice and is likely, and almost sure, to make a diagnosis on evidence that is insufficient.

With good plates—the type of plate that Dr. George and Dr. Roberts demonstrated and with the findings they demonstrated—there can be little or no question as to the correctness of the diagnosis. But when we discuss the other shadows, the ones which are less distinct than the clear-cut ones which they show, they say I should disregard their findings and not consider them. This is similar to what one would say about exaggerated bronchial shadows or even the normal markings of the lung. In almost every case Dr. George or Dr. Roberts would demonstrate that there had been some other evidence, that is, deformity of the cap or deformity of the pyloric end of the stomach. These findings, combined with the gall-bladder findings, make a composite picture which enables one to make the diagnosis.

When one takes all of these things into consideration, the question as to whether the shadow shown is or is not the outline of the gall-bladder is of little significance. Dr. George has said that the normal gall-bladder will probably show sometimes. The only point at issue is, do we ever show normal gall-bladders? Are these indistinct shadows visualized normal gall-bladders? When these gall-bladders show more distinctly than the ones I have been so disturbed about, even though I do not have any other evidence, I must consider them of clinical significance.

Dr. Dudley Roberts, New York: It is true that Dr. George and I have spent a hectic afternoon with Dr. Cole, but it is with some satisfaction that we all report an agreement on the essentials. It is true that as more detail is secured in skiagraphs of the upper abdomen interpretation of all the shadows is an extremely laborious and difficult undertaking. In the end we all agreed that the shadows that Dr. Cole regarded as somewhat suggestive and extremely confusing were not to be interpreted as gall-bladder shadows. I must at this time repeat that we cannot hope to visualize a gall-bladder because it contains abnormal bile, but because its mass is increased above the normal. The normal sized gall-bladder, even though it is chronically inflamed as to its mucosa and somewhat thickened as to the entire wall, cannot be visualized ordinarily and if it is one cannot be certain that it is abnormal. Chronic cholecystitis eludes Roentgen diagnosis unless there is dilation or stones.

We owe Doctor George a debt of gratitude for so thoroughly and satisfactorily presenting the various lines of Roentgen evidence of gall-bladder lesions. We must, of course, recognize the limitations of this method of diagnosis, but, on the other hand, we must not underrate its value either alone or combined with clinical evidence in the positive or negative diagnosis of diseases of the gall-bladder.

DR. WHITE: There is just one aspect of the subject that we must get clearly in our mind from this discussion tonight. It is common for the clinician to ask, "Did you find any gall stones?" as if those were the whole story. We have been shown what a multiplicity of signs there are of a pathological gall-bladder. It is not a question of stones; but we have, starting with the stomach and bending back of the stomach, a deformity of the duodenal cap—a picking up and pulling out of the second portion of the duodenum—a bunching up in the right quadrant, of a lot of jejunum that ought not to be there—and a picking up of the transverse part of the colon.

CLINICAL NOTES ON AN INTESTINAL INFECTION WITH GIARDIA DUODENALIS (LAMBLIA DUODENALIS) (DAVAINE, 1875, AND STILES, 1902).

By Dr. John C. Hemmeter,

BALTIMORE, MD.

This organism is at present also known by the name of "Giardia." I have described it in the second volume of my work on the "Diseases of the Intestines," pages 542-545. Unfortunately the various observers in Europe who have described this protozoan have given it different names, and even classed it under different zoologic headings, so that at the time I first observed this infection in the human being, in May, 1902, there were so many designations for it that I found great difficulty in classifying, and I sent some of the fecal evacuation to Dr. Charles Wardell Stiles, then of the Bureau of Animal Industry in the United States Department of Public Health. Dr. Stiles definitely classed the organism which I sent him as "Lamblia Duodenalis," and published a short note on this case in the Washington Medical Annals for 1902.

The various names applied to this type species are given in my work on the "Diseases of the Intestines," Volume II, page 543, with illustrations on page 545, but the zoologic characters of giardia (lamblia) are given by Charles Wardell Stiles in a monograph entitled "The Rat in Its Relation to Public Health," published by the United States Department of Public Health in 1910, page 87; more recently by Kofoid and Christiansen.

Synonyms and illustrations of this peculiar protozoan infection are there given, as studied from the field mouse.

It is not asserted that my case was the first infection of this organism ever observed clinically, but it is the first case observed in America, and as I have observed two other cases since then, and two further cases have occurred in the practice of Dr. M. Gibson Porter, Roland Park, Baltimore, Md., it may be of use to further clinical observation to give the details of this infection as I have observed them.

The patient, a male child three years old, had suffered from abdominal pain and distention ever since it had been weaned.

The child was born in Crisfield, Md., otherwise of healthy parents. The attached is a copy of my clinical history of the first case observed.

The object of this report is chiefly to give an aid to the diagnosis and to recommend an effective treatment of the infection with this flagellate protozoan, which was first described by Lambl in 1859, as found in the feces of children. He then designated it as Cercomonas Intestinalis. One of the best descriptions from the zoologic viewpoint is by Grassi and Schewiakoff, which is found in *Zeitschr. f. wissenschaftl. Zoologie,* Bd. 46, 1888, S. 143, from whom most of the drawings by Stiles and E. Adolph Schmidt are taken; more recently by William C. Boeck.

The parasite is very common in the upper part of the small intestine of the common house mouse, the brown and the black rat, the field mouse, and occurs also in domesticated cats, dogs, as well as in sheep, rabbits and guinea pigs. Man becomes infected through the eating of food that has been soiled by the excrements of mice and rats, which contain the encysted stage of this organism. Experimental infections have been produced in mice and rabbits by Perroncito, and Stiles produced an infection in guinea pigs by feeding them with human feces that contained the encysted stages.

Stiles states that Grassi produced an infection on himself. It has been observed in people who were apparently in good health.

I have gained the impression that children become infected when they are left to crawl about on floors that are accessible to the animals above referred to. Its habitat is chiefly in the duodenum and jejunum, where they attach themselves by means of a sucker in the anterior part of the giardia (lamblia). In severe cases it may occur in the ileum. The adult giardia (lamblia) are rare in the colon, for if the peristalsis is not much exaggerated, the encysted stage is found in the feces, but if the peristalsis is accelerated and diarrhea ensues, as was the case in all three of my patients, the organisms have no time to encyst and the freely motile forms are found in the stool.

To observe the motion well, the stools have to be kept warm and they have to be examined on the warm stage of the microscope, because when they are mixed with a lot of other fecal contents they may be readily overlooked unless they are actively in motion. In the unstained stool it is only the movements of the flagellae which draws attention to the giardia (lamblia). The motion becomes slow at 50° centigrade, and a temperature above 52° C. or 0° C. is fatal to them.

My main reason for assuming that this infection of the human intestine occurs more often than is usually believed is based upon two well-known facts: First, that systematic stool examinations are not practiced as a routine in general practice, and, second, that many of the agents used to remedy diarrheas and dysenteries are destructive to this protozoan. I must remark here that opium and bismuth are not curative, but simply relieve the pain and diarrhea for a while. But all derivatives of tanic acid are more or less destructive to giardia (lamblia). The unfortunate part of this rather effective therapy is that children can stand these derivatives only in such small amounts as will not suffice to kill off all the giardia (lamblia) in a short treatment. I will return to the therapy later on.

Diagnosis.

In any case of diarrhea or dysentery, especially in children, characterized by very offensive stools, the feces ought to be examined microscopically and then the recognition depends upon obtaining motile forms of these little animals. They are pear shaped, about 15.5 to 16.5 m. long and 10 to 12.5 m. broad. Viewed laterally, the organism looks like a tiny tadpole, the sucker on the lateral aspect assuming the appearance of a gigantic mouth. This effect is brought about by a concavity of the anterior part of the body, which looks as if a semicircular piece were cut out of the pear-shaped body, but on viewing the ventral aspect it is found that this concavity is not a complete circle, but that the posterior circumference of the concavity is formed into a kidney shape by a projection which is directed forward. In the depth of this concavity, which is known as the peristoma, there are two oval nuclei. In well-stained specimens these nuclei are connected by a narrow band. They have the shape of tiny cysts or vesicles, which in very active specimens can be seen to contract and expand. This is a very rare observation, for the giardia (lamblia) has to remain quiet, at least in the anterior part of the body, in order to make possible the change of form in these two vesicles. Contraction of the anterior body of the giardia (lamblia) may compress the nuclei mechanically.

The protoplasm of the giardia (lamblia) contains other enclosures. There are no other nuclei or vesicles apparent. It is bounded by a delicate limiting membrane. One cannot recognize any orifices that could be termed anal or mouth openings. The organism moves by means of four pairs of flagellae. The anterior pair are located on either side of the sucker concavity, just in front of the two nuclei, as just described. The middle double pair is attached to the posterior part of the sucker concavity, just where the indentation occurs that gives it the kidney shape. The posterior pair occurs at the extreme end. The movement is very rapid. In the small intestine they attach themselves to the epithelium by suction.

The nutrition of the creature occurs by osmatic processes. In animals the entire mucosa of the duodenum and jejunum has been found densely occupied by attached giardia (lamblia).

Pathogenicity.—Perroncito describes a fatal disease in rabbits due to this protozoan. P. Cohnheim (*Deutsch. med. Woch.*, 1903, 248) declares that they are not pathogenic and that it is not necessary to treat these infections in the human being with germicides. But Cohnheim admits that their occurrence in the feces has diagnostic importance. Zabel (*"Megastoma Intestinale und andere Parasiten in den Zotten eines Magenkrebses," Archiv für Verdauungskrankheiten,* Vol. VII, p. 509) gives nine different authors who describe giardia (lamblia) as the cause of gastrointestinal diseases. Others, however, describe cases where it was found without causing any gastrointestinal symptoms. According to Leuckart (Leipzig, 1863), the pathogenetic idea is reversed; namely, the giardia (lamblia) do not cause a disease of the intestines, but they attach themselves to the intestinal mucosa because this mucosa is already diseased. In other words, the normal defense of the intestines being lost by previous infections or catarrhal conditions or abnormalities of secretion and peristalsis, the giardia (lamblia) find a favorable environment for their propagation. This view is disproved by the fact that children may recover very rapidly after six or eight days of effective treatment, never again to show another symptom, provided they are kept away from the original source of infection.

In my opinion, a pre-existing enteritis, or ulcer of the stomach or intestines, could not possibly be cured in so short a time, and if giardia (lamblia) are found in the feces of children affected with diarrheas, etc., they are the cause of it, not the consequence of it. At the same time, I do not wish it to be understood that a pre-existing disease could not form the conditions for a more favorable development of these flagellates.

In an article by Nichols in the *American Journal of Medical Sciences*, 1905, 23 cases of entozoic infection of the stomach are reported. Of these, two were cases of chronic gastritis; one was an ulcer of the stomach; 19 were carcinomas. This would favor the opinion that entozoa grow most readily in a stomach that is diseased, and especially afflicted with such diseases as destroy the secretion of hydrochloric acid.

Wasserthal, in "*Archiv für Verdauungskrankheiten*," Vol. 13, 1907, p. 259, speaks especially of the occurrence of flagellates in achylia gastrica. He does not strictly specify giardia (lamblia) intestinalis. It is presumed that what would affect flagellates in general would affect the giardia (lamblia) intestinalis also. Concerning this I have my doubts, for the biologic conditions of the environments which are most favorable to the growth of the different kinds of protozoa differ markedly when they are observed and studied in the laboratory under conditions that are as near as possible normal to their natural life. It is therefore not proven that the conditions favorable to the development of one protozoan in the human gastrointestinal tract may be favorable to the growth of another protozoan of a different species.

Concerning the reaction of the stomach observed in the various types it may be said that C. W. Stiles (*l. c.*) reports, page 86, that P. Schmidt's case was one that showed an amount of hydrochloric acid equal to 1 per cent.; this must be an error, because no human could ever show that amount of hydrochloric acid. Stiles probably means .1 of 1 per cent.

In my own three cases, one of which Stiles described on page 91, lc., as the first American infection with this organism observed in this country, the amount of hydrochloric acid found was free HCl 24. In the other two cases that I have studied in regard to this point, the gastric juice was normal. Dr. M. Gibson Porter, of Roland Park, Baltimore, Md., has four cases under observation at the present time. In none of these was the gastric juice examined. One child had pronounced signs and symptoms of acidosis with air hunger and vomiting. There was no diarrhea, but exceedingly large stools containing live giardia (lamblia). This child, from last reports, was improving under a treatment consisting of small doses of potassium iodide, bicarbonate of soda and milk of magnesia.

Of the four cases of Dr. M. G. Porter only two have diarrhea. An annoying symptom was bed wetting, but no giardia (lamblia) were found in the urine. One of the children that did not have any diarrhea and no other symptoms was 11 years old and showed millions of live giardia (lamblia) in the stools. It would appear from Dr. Porter's cases that the infection can exist with enormous numbers of giardia (lamblia) occurring in the stools, and yet no very urgent symptoms be present. That something is wrong, however, is proven by the fact that such children are brought to the physician. The cases of this physician are still under treatment and he at the present writing cannot tell whether the giardia (lamblia) can be made to disappear or not.*

It is of note to record that one enterprising clinician had the daring to infect a child that was free from parasites by giving it 250 c.c. of suspicious water to drink on two different occasions and causing diarrheas (Epstein, *Prager med. Wochenschrift*, 1893, pp. 38-40). The organism thus experimented with belonged to the Trichomonads.

Lieut.-Col. C. M. Wenyon,** who has examined thousands of soldiers for the British Government, has published an extensive study of this giardia (lamblia) in particular, and other protozoan parasitic infections of the human intestine; also investigated the pathogenicity and carrier question, as well as treatment.

RECOGNITION OF THE GIARDIA (LAMBLIA) INTESTINALIS IN STOOL PREPARATIONS.

If it is desirable to see the organisms in motion, it is advisable to keep the stool warm until it reaches the laboratory. The glass slide and the stage should also be kept warm. It is advisable to put a small piece of horse hair between the slide and the cover slip, so as to prevent mashing of the giardia (lamblia) by the pressure of the glass. Zabel recommends staining with a watery solution of methyl blue. I find that this stain does not enter the live and perfectly healthy giardia (lamblia) very readily. In order to obtain a methyl blue stain the stool has to be dried in the air and slightly warmed, but then all the other parts of the stool may accept the blue stain and the recognition of the giardia (lamblia) becomes difficult. Methyl blue does not stain the flagellae.

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*January 2, 1920, Dr. Porter has reported the disappearance of the giardia (lamblia) from the stools.

**Lieut.-Col. C. M. Wenyon, B. Sc., M. B. B. S., and Capt. T. W. O'Connor, M. R. C. S.: "Wenyon Human Intestinal Protozoa in the Near East." An inquiry into some problems affecting the spread and incidence of intestinal protozoal infections of British troops and natives in the Near East, with special reference to the carrier question. Diagnosis and treatment of Amebic Dysentery and an account of three new human intestinal protozoa.

In order to stain the body of the giardia (lamblia) it is preferable to use a diluted solution of iodine. This does not stain the flagellae, but, according to the concentration, it stains the organism either slightly yellow or mahogany brown. An addition of a 5 per cent. acetic acid solution preceding the iodine stain will bring out the flagellae and the nuclei.* A 10 per cent. solution of sodium bicarbonate was said by Grassi to bring out the flagellae. This has failed in my experience. But if the stool is made after carbohydrate free diet is given for several days to avoid the blue color caused by iodine whenever it meets with carbohydrate remnants, then the giardia (lamblia) will stain a mahogany brown with the iodine. Fixation and drying by heat do not produce satisfactory results. But if it is desirable to make out the length of the flagellae, this can be done by holding an air dried specimen of feces in the fumes of a 1 per cent. osmic acid solution for a minute.

Giardia (lamblia) are capable of very rapid movement, caused by active lashing of the flagellae, and at the same time capable of changing the shape of their entire bodies by protoplasmic contractions. Sometimes they describe circles or they may attach themselves either with the head end or the tail end and undergo pendulum-like movements of that end which is not attached. The changeability of the form of the body of the animal is surprising. It seems to have considerable force to turn up the tail end either touching its own back with its tail or touching the ventral side near the peristom. During all this time, the size and depth of the peristom is changing also, appearing sometimes as if the giardia (lamblia) were opening and closing the peristom, just like a fish opens and closes its mouth. A favorite motion of the little animal is to propel itself straight forward if the field is clear, but during this forward motion it rolls and twists itself toward one side. After the specimen has cooled down the movements become less and less, until eventually all motion stops. The last to move are the flagellae attached to the outer side of the peristom.

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*Dr. Charles E. Simon has had experience with fixation by hot Schaudinn solution ($HgCl_2$ and C_2H_6O) which proved satisfactory.

The flagellae attached to the tail end are used mostly as steering organs; but the giardia (lamblia) can also attach themselves by means of these caudal flagellae. The most anterior pair of the flagellae are for motion and the direction of their lashes is from below upward. They appear to be mostly in use when the animal desires to rise upward. We have already said there are two pairs of flagellae attached to the posterior edge of the peristom at a projection which gives to the peristom a kidney shape. The outer pair of these middle flagellae issuing from the projection I have spoken of, generally lash upward towards the back of the animal. Their function is locomotion. But the inner pair of these middle flagellae have only very fine and serpentine movements. They remind one of the movement of a corkscrew. They are the most difficult to see of all the flagellae, and when they are pasted against the body of the giardia (lamblia) they give the impression as if there were a groove on the ventral side of the creature. These are the last of the flagellae to keep up their corkscrew-like motion after the rest have ceased lashing. Grassi gives the length of the flagellae at 10-14 m.

It has been presumed that bacteria and small cocci are driven into the peristom by the peculiar motion of these ventral flagellae and that therefore they serve a purpose of nutrition. It is, however, not likely to feed on bacteria, etc., as its nutrition is by ormosis exclusively.

If an artificial current of warm water is produced between the cover slip and the slide, it is seen that the giardia (lamblia) are capable of swimming against the stream. Stiles and the other authors whom we quote always describe two nuclei and depict the two as being connected by a crossbar. I have seen numerous giardia (lamblia) in which two nuclei could be described as undergoing a state of division (by fission). Very extraordinary movements have been observed in the large nucleus when it is present alone. I have not personally observed a division of an adult giardia (lamblia) into two daughter forms, though Kofoid and Zabel describe this.*

According to Kofoid and Christiansen (*Univ. of Cal., Pub.*, Vol. XVI, Nos. 2 and 3, Nov. 19, 1915), the giardia (lamblia) from the field mouse has a complex neuromotor apparatus, according to which the flagellae pass anteriorally within the cytoplasm in a sweeping curve to the opposite side of the body, meaning opposite to the side from which they emerge, meeting its mate of the opposite side in a crossing designated as the "anterior chiasma," which is a node in the two fibers in the medium plane. The anterolateral flagellum arises from the external of two minute nodes called the left and right lateral blepharoplast; thence it passes in a sweeping curve through the anterior chiasm to the opposite side and runs then in the rim of the systostome parallel to the anterior peristom as a somewhat thicker strand, usually obscured by the peristom. But before it emerges from the body of the giardia (lamblia) as a free anterior lateral flagellum, it passes through another node called the "lateral basal granule" (see Fig. 1, p. 25, article by Kofoid and Christiansen, lc.). The postro-lateral flagellum arises also from the blepharoplast posterially in the cystoplasm close and parallel to the axostyle on its ventral side to about the level of the middle nucleus, then diverges from the axostyle to emerge from the cytoplasm as a free flagellum, at a point a little more than midway between the points of emergence of the anterolateral and posterior flagellae. The posterior flagellum springs from the posterior basal granule on the tip of the axostyle which represents its intracytoplasmic part fused with that of its mate. The free ventral flagellum has no prolonged intracytoplasmic part and cannot be traced to the blepharoplast. The flagellae are one and a half times the length of the body and in most specimens, as drawn by various authors, are abbreviated either as a result of the staining technique or for the purpose of saving space. The axostyle is a single organ actually located and extending along the ventral surface from near the center of the cytostome to the posterior tip of the creature, where it ends in a chromatic basal granule. It is denser than the surrounding cytoplasm, but less so than other parts of the neuromotor apparatus. It represents the intracytoplasmic part of the two posterior flagellae. At its anterior end the axostyle divides into two and each end bears two deeply stained blepharoplasts which are directly connected to the nucleus and centrosome and kryosome by the intranuclei rhizoplast. The elipsoidal blepharoplasts give rise to the antrolateral and postrolateral flagellae and also to the posterior flagellae by way of the axostyle. The connections of the free ventral flagellae with this internal neuromotor structure have not yet been demonstrated, but may be expected. The parabasal bodies described by Kofoid, lc., are two slender, tapering and deeply staining rods lying in the posterior half of the body dorsal to the axostyle. They are about 0.5 the transdiameter of the body; they lie parallel to each other and tend obliquely posterially either to the right or left of the medium plane. No chromatic fibers connecting the parobasal bodies with the axostyle have been observed.

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*This appearance has led some observers to the mistake that it was a real groove and that the giardia (lamblia) was in a stage of fission; that is, of dividing into two. I do not wish to deny that the giardia (lamblia) can divide in this way, but one must be exceedingly careful to ascertain that one is looking at the real groove and not at a false groove created by the flagellae. Kofoid and Christiansen, in their article entitled, "On Giardia Microti Sp. Nov., From the Meadow Mouse," and also Wm. C. Boeck in his article entitled, "Mitosis in Giardia Microti," give minute descriptions of this process of fission in giardia (lamblia).

The organelles (lc. Kofoid, Fig. A) of this simplest (from the standpoint of numbers only) possible multicellular organism consist of two nuclei (nuc.), two blepharoplasts (bleph.), from both of which arises one interconnecting neuromotor system joining all of the organelles, one axostyle (ax.), two parabasal bodies (par. b.) and one large ventral cup or cytostome (cyt.).

In the giardia (lamblia), as observed in human stools, only two nuclei, the cystostome, the peristom and the flagellae exterior, to the cytoplasm are visible. The other details described by Kofoid in his article on "Binary and Multiple Fission in Giardia Muris (Grassi)" are not visible in the giardia (lamblia) from the human intestine.

It should be added that Von Jaksch (*Wiener klin. Wochenschr.*, 88, No. 25) is of the opinion that some of the enclosures of these parasites are red blood corpuscles. Some of the illustrations of Von Jaksch and Zabel justify the conclusion that they saw some of the forms of giardia (lamblia) described by Stiles, Kofoid and Boeck. The important point to bear in mind is that the enclosures of red blood corpuscles would signify a direct parasitic character to these creatures. This lends a new aspect to the clinical study of these protozoic infections and one not in harmony with the experience of those clinicians who describe millions of giardia (lamblia) in the stools of children that are not suffering from diarrheas and who are apparently not pathologically affected. Much must be left to future investigations of these conditions.

It appears that healthy individuals can stand the infection for six months, sometimes a year; in fact, it is very hard to ascertain how long the individual has had the parasites when the case first comes under clinical observation. But cases have been known to remain in fairly good health for six months, although the stool contained millions of giardia (lamblia).

In the practice of Dr. M. G. Porter, of Roland Park, Baltimore, Md., two little girls were the infected patients in the same family. One child suffered with diarrhea and the other was in apparent good health, although her stools, according to Dr. C. E. Simon's examination, contained the giardia (lamblia) in enormous numbers. One of the characteristics of the stools of giardia (lamblia) infection is their enormous size during the period that the patient is not afflicted with diarrhea. These copious evacuations are due to two facts: First, the parasites themselves may multiply to such an extent that they increase the bulk of the stool by their own numbers; they also prevent the normal bacteriocidal mechanism of the intestines from destroying the other microbes of the intestines, and there is therefore frequently a luxurious bacterial growth, evident in the feces. Secondly, the enormous number of giardia (lamblia) may cover the intestinal absorptive epithelium so completely that absorption is very seriously interfered with, and therefore the food cannot be reduced in bulk.

The acidosis which is sometimes observed is, in my opinion, due to slow starvation.

The absorptive power of the stomach was tested in my cases with the potassium iodide test and the reaction occurred in the saliva in ten minutes in both cases; but when the potassium iodide was introduced through a long duodenal (intestinal) tube, so that the end was demonstrable by radiography in the beginning of the ileum, there was no positive reaction in the saliva after three hours in one case, and none after six and one-half hours in another case. This does not necessarily mean that the epithelium was literally covered with giardia (lamblia), but it is conceivable that the mucosa was diseased before the giardia (lamblia) took up their habitat in that part of the intestine.

Some authors, like Zabel (*l. c.*), interpret the occurrence of giardia (lamblia) in the feces as always meaning that the stomach or the intestines are diseased. Some wish to have it understood that important conclusions as to the seat of the gastrointestinal infection could be made from the presence of these parasites in the stool.

Some of the cases of Dr. M. G. Porter had absolutely no gastrointestinal symptoms, and yet the stools, according to Dr. Charles E. Simon, contained billions of giardia (lamblia). In such cases, without symptoms, the presence of the giardia (lamblia) in the feces should always compel analysis of the gastric juice and extended tests of the intestinal functions, for it does not appear rational that such a luxurious growth of these protozoa could occur where the gastrointestinal apparatus is normal. It is possible that the symptoms are only latent in an otherwise healthy individual. Or we may have the following condition of affairs: the individual who shows the presence of giardia (lamblia) in the feces without symptoms has previously had some gastrointestinal disease, and not entirely recovered from it. Thereafter the parasite finds a lessened resistance of the alimentary tract to its growth.

In all of my cases the giardia (lamblia) or megastomas were associated with other protozoa and other parasites. I found numerous cocci and bacteria of different kinds. Transiently the stool contained spirilli; different kinds of leptothrix; amebae; trichomonads; cercemonads. Some of the specimens of stool were so filled with leptothrix and spirilli that they concealed the giardia (lamblia).

Von Jaksch (*Wien. klin. Wochenschr.*, 88, No. 25) described an animal parasite in the feces of children which for some time were thought to be products of the cells that line the stomach and intestines, but they were later identified as living protozoa and not products of the human cells. I have seen a number of these forms in my cases. As they are not yet classified, I must refer to Von Jaksch's article for their description. They are large, spherical formations that have at one part of the peripheri a semilunar excision. The organisms are homogeneous throughout, except the semilunar, which is darker. They do not show any flagellae. The organism is contractile, except that portion which I have called the semilunar excision. Should the creature turn so that this portion which is called the semilunar excision comes on top, it is found not to be semilunar, but a circle. Some of these forms may contain white and red blood cells within their interior; their dimension is six times as large as the diameter of a red corpuscle; they are not to be confounded with cercemonas intestinalis or large amebae; they have no resemblance to any intestinal infusorium that has come under my observation; they are undoubtedly infusoria of a type that has as yet not been classified. Von Jaksch and Zabel have not given it a name.

In all of the instances in which it has been observed, the patient suffered with dysentery.

The presence of red corpuscles within these unknown infusoria brings the thought that I should explain why no red corpuscles could be observed within the interior of giardia (lamblia) intestinalis. The principal reason is that the giardia (lamblia) is not large enough to contain a red corpuscle; its tail end is entirely too small to harbor a r. b. ç., which is at least 8 m. in diameter, whilst the anterior end of the giardia (lamblia) may be 5 to 15 m. long and 1 broad; the peristoma projects into the body of the giardia (lamblia) in a concave fashion (see Figs. 1, 2 and 4), so that no room would be left to be occupied by a full-sized red corpuscle; then, again, the anterior part of the body of the giardia (lamblia) contains one or more large nuclei. It is, however, well known from the biologic standpoint that the giardia (lamblia) carry on their nutrition by osmosis and do not take in the cells of the blood stream. The greatest divergence of opinion concerning pathogenicity is found in a discussion in the *Southern Med. Jour.* for January, 1920, page 23. In the discussion of a paper by Dr. John Jelks, one considers the flagellate infection more serious than amebiasis, whilst another states that in most tropical countries flagellates are normal inhabitants of the intestinal tract. Still another finds flagellates in 25 per cent. of the inhabitants of the Carolinas. A fourth states that it is possible to get rid of the giardia (lamblia) in a few days. All of these views I find difficult to harmonize with my experience. Lambliasis is not near as dangerous a condition as amebiasis; but, on the other hand, it is not to be taken too lightly, for even if the individual showing giardia (lamblia) in the stools is not severely ill, he may become so at any time if not treated.

As I happen to have been the first clinician that observed an infection with giardia (lamblia) intestinalis in America (first American case of infection with giardia (lamblia) intestinalis (Hemmeter's case) described by Charles Wardell Stiles in *Washington Medical Annals*, 1902), I have always been on the lookout for other cases of this kind.

The increased frequency with which protozoic infections of the human gastro-intestinal tract are observed during the year 1919 has suggested to me the thought that possibly our homecoming troops, nurses and other accompanying agents of a large army, are bringing these parasites with them. The fact that a number of the patients are children does not contradict this idea, for in a few local cases the children appear to have been infected from a nurse that had been in Constantinople.

The long persistence of live giardia (lamblia) in the feces, even after active treatment is undertaken, finds explanation in certain (1) biologic factors of the parasite; (2) anatomic conditions in the infected patient; (3) lack of a rational system of therapy based on the life habits and reactions of the parasites; (4) the unrecognized extrinsic and intrinsic sources of continual reinfection; (5) the impossibility to employ antiseptic substances within the intestine which outside of the body are known to quickly destroy the giardia (lamblia). This includes even comparatively harmless agents, that is, harmless for adults; for example, the ipecacuanha and its components, nicotine and ematine in sufficient strength to destroy the giardia (lamblia) cannot be employed in children; (6) not continuing the therapy long enough; the treatment must in all cases be continued for a long time and the feces examined microscopically once every week for a month after the disappearance of the parasites from the stool. The cysts cannot be destroyed by any kind of feasible therapy within the intestinal canal, but as they exist almost exclusively in the colon, they are not so difficult to get rid of, in my experience by colon

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NOTE: The title of the paper on which this discussion occurs is "The Prevalence of Ameba, Cercemonas Intestinalis-Hominis, and Pellagrous Infections in the South—The Responsibility Resting on Nation and State: Suggestions as to Means of Control." (*So. Med. Journ.*, January, 1920.)

lavage. The two most effective agents used for this purpose, and which can do no harm to the patient, are ichthyol, 1 drachm to 1 pint of warm water, or tannic acid, 30 grains to 1 pint of water. Carbolic acid should not be used in the human colon in any strength whatsoever; it is rapidly absorbed and produces general toxic symptoms, which are preceded by vertigo and buzzing in the ears. Grassi claims to have had success with calcined magnesia, and Dr. M. G. Porter, of Baltimore, has had favorable results with bicarbonate of soda and magnesia.

As there is a fairly unanimous opinion that the HCl acid of the gastric juice is detrimental to giardia (lamblia), the success of this alkaline treatment is paradoxical, unless the children are suffering from acidosis. The relation of this acidosis to lambliasis is obscure, but when it occurs in undernourished patients it is most probably due to incipient starvation. The intestine as a rule does not tolerate alkalies well, unless they are given to counteract a definitely ascertained excessive secretion of gastric HCl or, in rare cases, the hyperacidity due to the formation of organic acids from carbohydrate fermentation. At any rate, alkalies can only be permissible after a previous chemical analysis of the gastric contents, feces, blood and urine; but under no conditions should the HCl or the gastric juice be completely neutralized.

My best results were obtained when I could utilize the bile of the patient as a means of destroying the parasite. Normal bile cannot accomplish this; it is necessary to make the bile the vehicle of an antiseptic agent. The most effective agent was found to be hexamethylenamin tetramine (Urotropin). Six grams of menthol and six grams of hexamethylenamin are capable of rendering the bile sterile in infected gall-bladder cases, as I have found by making cultures from the bile, which was obtained by duodenal intubation. (See also J. Crowe, *J. H. H. Bulletin,* 1908, XIX, No. 205).

In children the dose has to be adapted to the age and weight of the child. I have found that 1 gr. of hexamethylenamin and ½ gr. of salol three times a day in a child three years old cleared the intestine of adult giardia (lamblia) after ten days of treatment. After these medicines have been given for three or four days I give a day's rest, on which castor oil is administered in the form of the aromatic castor oil of the National Formulary (see *Jour. A. M. A.,* Nov. 29, 1919, p. 1698).

Saccharin itself has a destructive influence upon the giardia (lamblia), and the amount contained in the *oleum ricini aromat N. F.* is sufficient to affect the parasite, though not efficient to kill it entirely.

Judging from the effect of the Roentgen rays on living and active specimens of giardia (lamblia) exposed to this form of ray, I should predict that the X-ray as well as the radium emanation could be used to stun these parasites, if not kill them. The parasite, when once severely injured, can be expelled by castor oil repeatedly given.

I have not treated any patients with radium or X-rays, but I would recommend that this method be given a trial.

All observers agree on the great sensitiveness of giardia (lamblia) to cold. This would suggest the treatment of the upper intestine by the injection of ice water through a duodenal tube, which would, however, be hazardous in children. I tried it in an adult on one occasion for another form of parasite, after which the patient, a man 28 years old, suffered from unpleasant enteralgia; still, a period of enteralgia not exceeding one hour would be worth while bearing if simple ice water, followed by a purge, would be sufficient to clear the intestine of these unwelcome intruders.

The diet, during all treatment, has to be adapted to the individual. In children, no hard and fixed rules can be established, though it is imperative that the nutrition of the child must be maintained. Any malnutrition may lead to acidosis.

All forms of food that can be made from milk, flour and eggs are permissible, particularly if the child takes them with enjoyment. In enfeebled children small doses of Elixir of Calisaya, with minute doses of strychnine, and sometimes stimulation of wine gelatine, hot tea or coffee are called for.

In the stools of lambliasis the giardia (lamblia) are not found in pure culture. There are, as a rule, other protozoa present, especially amebae and trichomonads. Sometimes leptothrix threads, spirillae and various monads are found together with transitional forms from the freely movable adult stage to the encysted stage of the giardia (lamblia). Some of the cysts when evacuated by the embryo giardia (lamblia) present a helmet or cup-shaped appearance. In some of the very young giardia (lamblia) the flagellae are not recognizable.

In microscopic examinations with an oil immersion lense the giardia (lamblia) are found mixed with food detritus, fragments of the epithelial lining of the small and large intestines, rectum, and sometimes of the stomach, especially if the stomach has been aspirated with a stomach tube and the giardia (lamblia) found in the stomach contents. The microscopic picture in severe cases of lambliasis, as thus found, is, as a rule, a very complex one; not only are other protozoa and many kinds of bacteria present, but the transitional forms may assume almost any conceivable appearance, so that the only safeguard in recognizing the giardia (lamblia) is a thorough familiarity with their normal appearance. To aid this the author has added a number of pen and ink sketches of this organism in the various attitudes it may assume, and also a drawing of a typical picture as seen with an oil immersion (A. Leitz, 1 15).

THE TREATMENT OF GIARDIASIS OR LAMBLIASIS.

Wenyon (l.c.) gives a rational explanation of the tendency of giardiasis (lambliasis) to return. In examining a number of animals infected with these protozoan he found that these flagellates live not only on the surface of the intestines, but many of them make their way into the glands, where they can be seen in rows on the glandular epithelium. It is possible that they may spread into the bile duct, but there is as yet no definite investigation of this subject. The important point in the treatment, and one which makes it so difficult, is that the intestinal disinfectants act directly on these flagellates only in the lumen of the gut, but not on those within the glands. These escape destruction and eventually, when the treatment is stopped, re-establish the bowel infection. It is for this reason that I sought for a treatment which could attack the organisms more directly, not on the surface of the gut only, but by being absorbed, like the bile salts from the lower intestine, could pass through the portal circulation, enter the bile as it is secreted, and re-enter the intestinal canal with the expulsion of the bile. While such drugs should be disinfectant, they should not, in the therapeutic doses, affect the tissues and the household of the individual to whom they are administered.

According to Brauer, methyl blue (*Zeitschr. f. physiol. Chem.*, 1904, Bd. 40) when injected subcutaneously, or administered by the intestines, always appears anew in the bile and is then re-absorbed from the intestines, and although a part of the methyl blue is excreted by the urine, it takes a very long time for its final elimination. Similarly, menthol and hexamethylenamin undergo this singular process of absorption from the intestine, passage through the portal circulation, expulsion into the intestine with the bile and re-absorption. It is impossible to say how long it will take a single dose of hexamethylenamin to be eliminated entirely; much depends upon the sufficiency of the renal excretion and the oxidative powers of the organism. Six grams menthol and five grams of hexamethylenamin in the day can render a bile infected with typhoid bacilli and streptococci sterile (J. Crowe, *J. H. H. Hosp. Bull.*, 1908, No. 205).

Treatment Through the Duodenal or Intestinal Tube.

The intestinal tube has been introduced through the entire length of the small intestine by Einhorn, and on one occasion, when this clinician attempted to withdraw the tube, it broke off after seven feet had pulled out through the mouth. The intestinal tube is nothing but a rubber catheter about 15 feet long, which has to be introduced with caution and very slowly. It is not necessary to introduce the tube as far as Einhorn did; the duodenal tube devised by various clinicians will answer very well if it is about two meters long. There are markings on the Einhorn tube made of lead and asphalt, so as to make them visible by X-rays after the tube has been swallowed; but the tube itself is visible by the X-rays.

Dr. Jacob Buckstein, in August, 1919, *J. A. M. A.*, 73, page 670; also *J. A. M. A.*, March 6, 1920, has described an intestinal tube of his own invention which enhances the usefulness of the duodenal in intestinal feeding and intestinal therapy. The shifting motion of the intestinal coils, as indicated by the changing outline of the rubber tube, throws some light on the mechanics of peristalsis, and on the therapeutic value of exercise in constipation. This tube makes it possible to introduce therapeutic agents at specific portions of the intestines. The secretions on different portions of the intestines below the duodenum may be obtained for the purpose of biochemical and bacteriologic examination.

Details of Intestinal Tube Therapy.

After the tube has reached the duodenum, this is carefully washed out by repeated injections and aspirations of a solution of dilute HCl about the strength of normal gastric juice (2 to 3 parts per M.). If giardia (lamblia) are abundant in this part of the intestine, the lavage should be continued until the water comes back free of the parasite. Now the tube is allowed to pass to a distance of about two feet below the pylorus; then 1 gram or 15 gr. of hexamethylenamin is injected, dissolved in a solution of HCl, one part to the thousand. It must be remembered that alkalies check the dissociation of hexamethylenamin and therefore alkalies must be avoided during this treatment, forthe action of the hexamethylenamin depends upon the liberation of formaldehyde, which is more energetic in an acid environment. There are other preparations of this substance which can be used; for example, urotropin or helmitol, which is anhydromethylencitrate urotropin; also hippol, which is methylenhippuric. Hippol has the advantage of being dissociated even in an alkaline medium. I give the hexamethylen injections, not into the duodenum, but about one foot below the duodenum. Even when it is introduced through a colon tube into the ascending colon it can be detected as formaldehyde in the bile when this is aspirated through the duodenal tube one hour afterward.

This treatment must be alternated by injections of salol, 5 gr. three times a day for an adult, suspended in warm water. Every three days the duodenum must be washed out with warm normal salt solution and the entire colon irrigated with a solution of tanic acid, 60 gr. to the quart of hot water (4 grams tanic acid to the liter). If there are indications of colitis (and these are present in almost all cases), the healing of the colon is promoted by irrigating with a suspension of aluminum silicate, 50 grams to 1,500 c.c. of water. If the patient can drink this aluminum silicate in a manner which is followed when a barium carbonate meal is given for radiographic purposes, it will arrest the diarrhea and mechanically remove millions of giardia (lamblia). The objection to the aluminum silicate treatment (kaolin) is that it is very bulky to swallow, but it can, as a rule, be taken by patients determined to get well.

Methylene blue, which is mentioned with the technique for staining the giardia (lamblia) in the stools, has been found an effective agent for the destruction of these parasites. METHYL BLUE IS A SALT OF THE BASE TETRAMETHYLTHIONIN. It occurs in two forms: (1) the methyl blue chlorohydrate HCl $(C\,H_3)_2$ N S N (CH3)$_2$; and (2) as the zinc chloride double salt 2 $(C_{16}H_{18}N_3SCl)$ Zn Cl_2H_2O. Following the example of C. Fairise and L. Jannin, *"Dyssent. Chronic à Lamblia, Archiv. de Méd. Exp. et d'anat pathol.,"* T. 25, No. 5, p. 525, I have used colon irrigations with a 1 per cent. methyl blue solution and also injected 50 c.c. of ½ per cent. of methyl blue through the duodenal tube. The results were so gratifying that I shall try again in the very first case that presents itself.

In order to judge the effectiveness of methyl blue in this form of protozoic infection, it is necessary to avoid all other treatment and use no other chemical substance during the period of methyl blue treatment.

In all cases where amebiasis is associated with lambliasis emetin treatment must be given simultaneously with the treatment direct against the giardia (lamblia). Col. Wenyon believes that injections of 1 gr. a day for 12 days sometimes causes the lamblia infection to disappear. This is true both when it is given by the mouth and also when it is injected hyperdermically; but practically in all cases where emetin is used the infection returns later on. In one of Wenyon's cases there was a control of 40 days after the treatment during which examinations of the stools did not evidence a recurrence of the parasite. This case, however, was given a course of 15 gr. of beta-naphthol with bismuth salicylate 20 gr. three times a day for 12 days after the course of emetin was finished. Wenyon states that beta-naphthol alone will not cause a disappearance of the parasite.

B. Naphthol and Bismuth Salicylate.—According to Wenyon, these two drugs in the form of a powder in the proportions just mentioned above have been given to a number of cases of giardia (lamblia) infection. In all these the infection has disappeared after a varying number of days, sometimes after two days' treatment, at other times after seven or eight days. So constant has been the disappearance of giardia (lamblia) cysts from the stools of cases that it is difficult to believe that the drug has no action on the infection and that the vanishing of the cysts is the result of the natural course of the infection. Unfortunately, however, the cure is only a temporary one, as the infection almost invariably reappears after a week or two of control. Only in the single case mentioned on page 128 under the emetin treatment did it appear that a cure has been effected, but whether this was the result of the emetin or the subsequent treatment cannot be stated.

It should be added, however, that bismuth salicylate when used alone is just as certain as when the beta-naphthol is used with it. It will usually cause a giardia (lamblia) infection to disappear, for a while at least. Everyone who has treated giardia (lamblia) infections or amebiasis, or both associated in the same individual, must be aware that the symptoms are, as a rule, intermittent and that sometimes they subside without any treatment whatever. There are periods when the giardiasis or lamblia infection is in abeyance, as judged by the cysts in the stools. I have a case under observation at present—a soldier who had been in camp in Texas. For the first week of the observation both giardia (lamblia) as well as amebae hemolytica were present in the stools. At the end of the seventh day, without any special treatment, except dieting, the giardia (lamblia) disappeared from the stool, but the amebae continued. During the second week he received treatment by injections of emetin hydrochloride and washing out of the colon with a tanic acid solution. At the end of the third week the amebae had disappeared. At the end of the fourth week giardia (lamblia) cysts made their appearance again in the stools. The treatment is not complete, for I intend to begin a treatment with hexamethylenamin through the duodenal tube.

No hard and fast routine treatment can be outlined. The physical and psychic state of the patient sometimes demand a period of rest without any washings of the duodenum or colon, but simply giving emetin hyperdermically and bismuth salicylate by the mouth. Cases that are not run down can be at once treated by duodenal injections of hexamethylenamin, together with lavage of the colon with a weak tannic acid solution. After a week of this, lavage of the duodenum as well as the colon should be stopped and the patient put on bismuth salicylate for three or four days, the main object of this being to give a few days of rest. Then the treatment by the duodenal tube as well as the colon irrigation should be resumed.

When patients of this type are persuaded to come back to the clinician once in three or four weeks, it is found that some are cured and some are not. This does not only depend upon the general physical state of the patient nor upon the invasion of the intestinal glands by the parasite, but I believe that there are varying degrees of virulence. Some giardia (lamblia) appear to be particularly vicious and resist the treatment even in patients that are physically not run down, but persistence on the part of the patient and clinician will be rewarded by a cure if the intubation treatment is repeated for a week every month after the supposed cure has been accomplished. Six months of this kind of control in very bad cases is the desideratum. C. Fairise and C. Jacquot, (*"Colite Ulcereuse Due a un Parasite Flagelle le Lamblia Intestinalis." Perforations Puis Occlusion Intestinale * Mort * Etude des Lesions Produites par le Parasite, Archiv. des Mal de* 1 *App. Digest et de la Nutrit,* T7, No. 6, pp. 301-317, 1913), have observed a case of ulcerated colitis due to giardia (lamblia) infection in which a perforation intestinal occlusion and death occurred. The patient was operated for the intestinal occlusion, but died six months later, giardia (lamblia) being found in the stools and free in the peritoneum after death. This case illustrates what giardia (lamblia) may do if permitted to continue their infection. It also explains the divergence of opinion in a symposium before the Southern Med. Assn. already referred to. Some cases may be very slight and not affect the general health; other cases may prove fatal. Whenever both giardia (lamblia) and amebae hemolithica are present, the case should be considered very serious, put to bed, and treated according to the methods outlined for infection with both of these parasites.

DISCUSSION.

Dr. S. K. Simon, New Orleans, La.: I think that the Association is to be considered fortunate in that Dr. Hemmeter has brought this matter of protozoal diseases before the Association, and that he has taken up the question of this particular protozoan. I also believe that we are to be considered lucky that he has brought this matter before us today, because there are one or two things that should be stressed. One is that our former conception that these infections are all caused by tropical or subtropical organisms has been proven to be fallacious. The protozoa, while they have their principal habitat in tropical countries, have, owing to the increased facilities for travel, become scattered throughout the entire world. The Spanish-American War was probably the occasion for the profuse spreading of protozoal infections in this country. Our soldiers came back from the countries south of us carrying amoebae of different varieties.

As far as the distribution of giardia intestinalis is concerned, there is considerable difference of opinion. So far as my experience goes, giardia is rather rare in the Gulf Region of the South. Dr. Hemmeter is wrong about the organism discussed at the meeting of the Southern Medical Association; it was the Balantidium coli and not the giardia intestinalis. In that regard, too, there seems to be a difference of opinion.

Dr. Hemmeter's series of cases in Baltimore is a rather notable one—12 cases in his experience, and shows the rarity of this infection in the United States. Sanford and White, years ago, examined stools, and found in them only six instances of giardia infection.

As far as morphology is concerned, I have myself seen only five instances of giardia infection in a large number of stools in cases of infection from tropical countries that I have examined. This infection could not be mistaken for any other organism. It has eight flagella, and swims around with a peculiar swaying movement. Its parastoma is not a mouth, but a suctile organ, by means of which it attaches itself very intimately to the intestinal wall. Of all the flagellates, the giardia has the best claim to pathogenicity; because it has a means of attaching itself to the intestinal wall and burrowing down into the glands, which the others do not seem to be able to do. Dr. Stiles says that in his experience in the tropics, a very large percentage of the intractable diarrheas were due to this organism. Many authors, however, believe that giardia is not pathogenic; that, in common with other flagella, it is merely a secondary invader of the intestinal tract, and appears in increased numbers only when the stools are loose; and that the diarrhea is, in fact, due to other conditions, and not to the presence of the flagellates. He thinks that the latter cannot start the diarrhea, but may increase it by their whiplike movements and the mechanical insult that they are capable of inflicting on the intestinal mucosa. That is still a matter to be decided.

One further point to be considered is that as all intestinal protozoa pass through two stages, the vegetative and the cystic stage. In the vegetative stage they do not usually appear in the stools; but if you give the patient a good dose of salts, so as to produce a loose, full movement, you find the vegetative forms in the stools.

Now one word regarding treatment. Dr. Hemmeter's suggestion is a most excellent one. The treatment of giardia infection, up to the present time, has been almost hopeless. Ipecac and emetin have been found to have no effect on any of the flagellates; and I shall, during the coming year, put Dr. Hemmeter's idea into active use, not only in giardia infection, but also in infection with Trypanosoma intestinalis, which we always have with us.

DR. FRANK SMITHIES, Chicago, Ill.: About four years ago, I reported observations on one thousand stool examinations; and in that collection of cases, we had three instances of giardia in individuals who had never been outside of the Temperate Zone. I wish to emphasize certain facts regarding recognition and treatment of this condition: first, collection of the stools. Remember that the infection occurs primarily in the upper alimentary tract, and that examination of the rectal mucosa for so-called rectal ulcer, or examining the stools from the colon, will not get the giardia intestinalis. One must have the patient in a state of free catharsis by the use of salines and collect the feces from the upper alimentary tract. One may have to search diligently, week after week, before he finds the parasite. We have reported instances of removed gall-bladders in which we found Giardiae, Trichomonas, Cercomonas and Endomebae histolyticae. Therefore, ascending duct infection does occur. We examined everything by means of the high power. I am selfish in doing this, because I hate to feel that someone is 200 diameters ahead of me. A low-power observation is absolutely worthless with respect to differentiation of protozoa.

I have cured three cases of giardiae. These were not merely improved but cured. Those who know me know that I do not say I cure many kinds of disease; indeed, I am from Missouri, when it comes to curing diseases. However, I have cured three cases of giardiae; and they have remained cured between three and four years. In instituting therapy, I put patients on a fluid diet for three days. At the end of the third day I give three doses of thymol in capsules in honey at hour intervals. Forty grains of thymol is administered at each dose. The medicine is usually given only in the evening. The patient is then kept food-free for eighteen hours, receiving nothing but black coffee. At the end of nine hours he gets two ounces of Epsom salts. By this means, I secure thymol saturation of the entire intestinal tract. If there is any pain, a small dose of opium takes care of this.

The above routine may require repetition several times at five or six day intervals. The examination of the stools is the only criterion by which we can judge whether or no an individual is free from the parasite. The warm stool must be examined for three consecutive days. We look especially for cysts. So long as individuals have encysted forms, and so long as we find these encysted forms in the stools, treatment must be repeated. For the infection in the large bowel, we give, twice a day, an irrigation of quinine and thymol. (Thymol 1:3000; quinine 1:1000; quantity, 3 liters.) It is usually well borne. We have infected people come back four times every year for careful stool study, even after cysts have disappeared from the freshly passed stools examined on a warm microscope stage. If repeated examinations disclose encysted forms found, we repeat the treatment. We have found that the gall-bladder is a favorable site for the retention of cysts as well as actively viable organisms. I have used methylene blue and practically everything else, but have found that the intensive thymol treatment with irrigations has proved very efficacious. Occasionally we have used calomel, with definite results when the predominating organism is a flagellate.

DR. W. A. BASTEDO, New York City: There is one point in the therapeutics that I wish to refer to. Dr. Hemmeter laid stress on the use of urotropin as a discharger of formaldehyde in the gall-bladder and intestine, but many investigators have shown that this drug does not give off formaldehyde in the gall-bladder or the intestine. In tests that are absolutely positive in dilutions of 1:150,000 of formaldehyde, Burnam found that when urotropin was added to the bile or intestinal contents absolutely no formaldehyde was formed; and only the merest traces or none at all in the gall-bladder or intestines when the drug was given by mouth in the largest doses that could be given. It is useless as a discharger of formaldehyde in any but acid liquids.

DR. BASTEDO (closing): With regard to Dr. Deaver's statement, I would say to Dr. Hemmeter that it was published in the *Annals of Surgery*, Vol. 65, page 247, under the Proceedings of the Philadelphia Academy of Surgery. In regard to nervous influences, I would record the interesting experience that some years ago, when we were experimenting with dogs with gastric fistulae in order to obtain gastric juice, we noted that when the laboratory man who took care of the dogs was cross when he came in the morning, and said to the dogs, "Get over there," when he cleaned their cages, we would not get any flow of gastric juice; whereas if he was in a good humor and patted the dogs on the back, and they wagged their tails, we got a copious flow. So I always say that good digestion depends on how we wag our tails. I do not need to elaborate on that.

DR. HEMMETER (closing): I had not known of the observations of Dr. Smithies concerning infections of the gall-bladder with giardia.

DR. SMITHIES: The paper is published in the *American Journal of the Medical Sciences* of four years ago.

DR. HEMMETER: Those familiar with the chemistry of formaldehyde know that none of the tests ordinarily used will show formaldehyde as formaldehyde, owing to the property that Aldehydes have of doubling up with other compounds. That does not mean that it could not be given off slowly and in quantity sufficient to affect a delicate flagellate like this. I am not able to deny the statements that Dr. Bastedo has made. But my method does not depend on the disinfection of the gall-bladder by urotropin, etc., but rather upon the successive lavage of the stomach, duodenum and ileum by the duodenal tube, using ichthyol, tannic acid, urotropin, as indicated.

DR. BASTEDO: It does not sterilize the bile.

DR. HEMMETER: I am not prepared to deny that. In addition to that, we gave salol. The mechanical removal of the protozoa by means of continual and progressive lavage of the intestine as far as can be reached in the colon and the small intestine is a great aid to the treatment with thymol in the gall-bladder and duodenum. Some of the gall-bladders are so filled with stones or lipoid masses (described by W. C. McCarty in *Annals of Surgery*, February, 1919). That it is impossible for any bile to reach the gall-bladder, as disinfection implies direct contact of the disinfecting chemical with the infected organ, it is obvious that no compound of formaldehyde can sterilize the bile if it is not excreted through the liver by way of the gall-bladder.

ROENTGENOLOGICAL ASPECTS OF LOWER RIGHT QUADRANT LESIONS.

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The diagnosis of lower right quadrant lesions is a matter of great importance; not only is this because of the frequent occurrence of appendicitis but because of other lesions which are apt to present themselves in this region, the differential diagnosis of which is often fraught with much difficulty.

Among the affections most frequently observed in the lower right quadrant of the abdomen may be mentioned:

1. Appendicitis.
2. Incompetent ileocecal valve and ileal stasis.
3. Dilatation of the cecum with stasis.
4. Adhesions and angulations.
5. Ulcerations due to tuberculosis.
6. Ulcerations due to carcinoma.

In the Roentgen ray study of lower right quadrant lesions both bismuth meals and bismuth enemata must be employed. When the bismuth meal is taken it ordinarily reaches the cecum in 5 to 8 hours, according to the character of the meal, and the transverse colon in 12 hours. Delay in the passage of the bismuth may be due to a dilatation of the cecum, to ptosis or to adhesions, to ileal stasis and angulation; ulcerations or carcinoma. The bismuth enemata supplement the information obtained by the meal. These consist of 5 ounces of bismuth subcarbonate to the liter of water, to which sufficient mucilage of acacia is added to suspend the bismuth. The patient is placed in the knee-chest position, and the low enema is allowed to run in slowly by gravity. This method of examination is especially useful not only in determining lesions of the lower bowel but also of the lower right quadrant.

1. *Appendicitis. Acute Appendicitis.*—Pfahler has called attention to the diagnostic value of the Roentgen ray in acute appen-

dicitis in certain instances, in that in those cases in which symptoms of acute appendicitis appear, due to beginning pneumonia in the lower right lobe, the lesion can be demonstrated in the lung, thus differentiating pneumonia from appendicitis, and that by filling the colon and thus determining the area of acute tenderness acute appendicitis may be differentiated from other acute lesions in the lower right quadrant.

Chronic Appendicitis.—A Roentgen ray examination of the appendix itself in its chronic state is capable of rendering valuable service in many instances. This, of course, only applies to those cases in which the lumen is patent, while in the very chronic forms the lumen of the appendix may be completely closed as a result of an obliterative process, and consequently the bismuth will fail to enter and the appendix naturally cannot be visualized. Case recommends a special technic for the examination of the appendix, and emphasizes two points: (1) the necessity of examining the patient in a horizontal position, with the screen over the abdomen and the tube underneath the table; (2) the necessity of palpating the abdomen under the screen with the gloved finger or, preferably, with a wooden spoon, and noting the localized tenderness. The time of the examination is of some importance. In six hours usually the cecum is fairly filled, and at that time the appendix under palpation will also probably fill. From this time on until the bowels are empty, and frequently for some time afterwards, the appendix remains visible. The ordinary technic will frequently not show the appendix, so that we must first find the appendix fluoroscopically and make a Roentgenogram in the position in which the appendix is visible. When the appendix remains visible for more than a day or two after the bismuth examination it is, in proportion to its poor drainage, a dangerous appendix. There are certain instances in which the bismuth shadow may persist for days, and in one or two instances it has been seen for weeks after the bismuth examination.

When visualizing the appendix the tenderness can often be localized immediately over it, and if the appendix be moved by the hand or spoon the tenderness will move with it. When the appendix is retrocecal the cecum must be pushed aside to visualize the appendix. A great majority of these retrocecal appendices show tenderness upon direct palpation. In those cases in which there is no tenderness, appendicitis may usually be excluded, but if the cecum is adherent there is always a possibility that this is due to some inflammation of the appendix in the past, even if the appendix is visualized and not tender. Pfahler claims that if there is localized tenderness over a fixed cecum, even if the appendix is not visualized, it means a pathologic appendix. The frequency of the visualization of the appendix by means of the bismuth meal is a much disputed point, varying according to several authorities from 35 to 90 per cent. The writers, however, do not believe every visualized appendix is necessarily pathologic. An appendix can be frequently found well filled with bismuth, yet lying perfectly free, no signs of adhesions and the appendix emptying with the emptying of the cecum. Under certain conditions it seems there is no reason why the appendix should not fill without being pathologic. On the other hand, when we find an appendix filled with bismuth, curled up and fixed, this invariably means some pathologic process is present. When chronically inflamed an appendix is frequently adherent to the structures surrounding it. It may be fixed throughout its length or only partially so. It is a frequent occurrence to find the tip fixed with the body of the appendix lying free. If the appendix be palpated under these conditions, the tenderness would indicate the presence of the chronic inflammatory process. The kinking of the appendix, which remains constant, is, according to Pfahler, a significant sign of adhesions; and when the appendix constantly points upward toward the gall-bladder region there is good ground to believe it to be in a pathologic state. The writers' experiences in this particular case agree with the findings of Pfahler. Attention has been called to the fact that in cases of chronic appendicitis, when studied fluoroscopically and radiographically, the pyloric end of the stomach is frequently observed to be lying down in the lower right quadrant region. This is not due to adhesions, but probably to the omentum wandering toward the inflammatory process. In such instances the stomach, when viewed under the fluoroscopic screen, can be moved to its normal position and yet will immediately upon release of the hand return to the lower right quadrant region. The reason for this abnormal position cannot be explained, but is probably due to some chemotactic attraction and, when present, is a very definite indication of a pathologic appendix. At times, however, this position is due to adhesions from the appendix extending into the omentum, thus actually causing the stomach to be adherent in that position. We have frequently pointed out how partial obstructions of the pylorus, due to inflammatory conditions, are liable to lead to the production of adhesions extending to the lower right quadrant region, dragging the stomach in that direction, and thus the appendix may become secondarily affected. Adhesions associated with the appendix may, however, extend to the surrounding structures and may lead to varying degrees of cecal and ileal stasis, and even to partial colonic obstruction. While the Roentgen ray renders valuable service in the diagnosis in many instances of chronic appendicitis, there are a certain number of cases in which it does not demonstrate the condition or where its results may be misleading. This may be due to the fact that on account of its unusual position the appendix cannot be visualized or that its lumen is closed, due to an obliterative process, or for some reason the bismuth is prevented from entering, or there may be an absence of tenderness, especially between attacks. Both Carmen and Case have called attention to the fact that, although retention of the bismuth in the appendix in plates does not necessarily always point toward a pathologic condition, yet, on the other hand, the appendix may be markedly diseased without being visible in the plates. In cases of subacute appendicitis, on account of the inflamed state of the appendix, the meal frequently will not enter, and there may be no adhesions. Fluoroscopically and radiographically nothing abnormal can be demonstrated in this region. The only sign that can be elicited will be tenderness over the appendix region. This tenderness, however, is also seen in a chronically distended cecum, and consequently it is not always a diagnostic sign referring to the appendix alone.

Attention must also be called to a patent source of error which should always be borne in mind. As a reflex condition a chronic appendicitis may give rise to a picture somewhat similar to that observed in duodenal ulcer; that is, there is gastric and doudenal hypermotility, with a definite filling defect and deformity of the duodenal cap. If this picture is rather marked and the appendicular adhesions are slight, one can understand how a chronic appendicitis might be mistaken for a duodenal ulcer, and, in fact, this error is sometimes made. In instances of this kind the plates must be most carefully studied, repeated examinations made if necessary, and in most instances a combined fluoroscopic examination, study of the plates, together with the clinical history, will point to the correct diagnosis; but even at times after a most careful investigation in every direction the correct diagnosis may still remain in doubt.

2. *Incompetent Ileocecal Valve and Ileal Stasis.*—This condition is usually indicated by the fact that at the end of 24 hours after a bismuth meal the ileum is entirely empty, and yet at the end of 36 to 48 hours the terminal ileum is filled, indicating the presence of regurgitation from the cecum to the ileum, due to an incompetent ileocecal valve. According to Case, in about one-sixth of 3,000 persons examined, most of whom were constipated and all affected with gastrointestinal disturbances, the bismuth enema passed the ileocecal valve and filled the terminal ileum for certain distances. The ileocecal valve is normally competent, and evidence of incompetency of this valve is indicated by the reflux of the bismuth meal from the colon into the ileum. Case insists that Roentgenoscopic examination for this purpose must be conducted with the patient horizontal, so that accurate visualized palpation may be practiced under the fluorescent screen. Not infrequently, by means of massage in the antiperistaltic direction, bismuth may be forced back into the ileum from the cecum, which indicates marked incompetency.

His method of testing the competency of the valve is the following: "The patient should lie supine on the horizontal fluoroscopic table. It is not necessary to introduce the rectal point or colon tube more than one or two inches. The container should be placed not higher than two feet above the patient and the barium or bismuth enema allowed to flow by gravity, the course of the bismuth column being watched fluoroscopically. Ordinarily 1,200 c.c. of the barium enema at 100° F. will suffice to fill the colon. It is important that a uniform technic be followed. I insist on seeing, by means of the fluoroscope, that the cecum is well filled. Massage of the abdomen over the shadow of the cecum is practiced in the antiperistaltic direction. Still further to insure complete filling of the cecum the patient is sometimes asked to lie on the right side for 10 to 15 minutes after the injection of bismuth, and a second examination is made when the patient returns to the table after evacuating the colon. The Roentgenograms are made with the patient lying prone, plate anterior, rather than lying supine, plate anterior. In the latter position 'saddling' of the ileum over the iliopectineal line may lead to a confusion. In marked cases of ileocolic valve insufficiency, however, there is never any difficulty in recognizing the terminal ileum." When the bismuth meal is taken it collects in about four hours in the ileum, while within eight or nine hours the ileum is normally empty of its contents. Delay in its passage may be occasioned by spasm incompetency of the ileocecal valve, bands of adhesions, displacements, prolapse or tumors; dilatation of the terminal portion of the ileum points to obstruction. In the study of ileal stasis care should be exercised to determine the emptying time of the stomach, inasmuch as any delay in the evacuation of the stomach may prevent the meal from reaching the ileum. When the bismuth is given by mouth and we find an actual regurgitation of the bismuth from the cecum back into the ileum, we are probably dealing with a true incompetency of the iliocecal valve. Incompetency, however, may be produced by means of the bismuth enemata if the enema is put in under too much pressure. When this is done, extremely violent antiperistaltic waves are set up which force the bismuth through the valve. The writers have repeatedly failed to demonstrate this incompetency by the bismuth meal, and yet incompetency could be produced by giving the enema under forced pressure, so that one must be very careful in drawing conclusions when the examination is made by means of enemata.

3. *Dilatation of the Cecum with Retention.*—When the bismuth meal is taken it ordinarily reaches the cecum in from seven to ten hours. Delay in the passage of the bismuth may be due to dilatation and retention in the cecum, which can easily be discovered by means of the Roentgen ray. The cecal stasis may in some instances be associated with a chronic appendicitis, and both may, as Brown has pointed out, be due to high degrees of enteroptosis as a result of a secondary low-grade inflammatory process. The degree of cecal stasis may vary markedly in various individuals, 24 or even 48 hour retention not being unusual in some individuals. While the Roentgen ray points out clearly the location of the cecum and colon, yet ptosis is a question of minor importance; similarly the position of adhesions and membrane attachments only play an important role in so far as they may lead to an interference with the function of the bowel. It is not the position of the cecum and colon with which we are most

concerned, but the functioning of this portion of the digestive tract; for directly in proportion to disturbances of function are symptoms of retention and partial obstruction produced.

The Roentgen ray examination of the dilated cecum has probably thrown more light upon this type of constipation than we have heretofore had. It is interesting to note that with a dilated cecum the patient may frequently not complain of constipation; on the contrary, state that the bowels move regularly every day. Yet in these cases one will find that a large part of the bismuth will remain in the cecum from 48 to 120 hours. The bismuth will become almost adherent in masses to the sides of the cecum and allowing only a small channel in the center. In some of the most obstinate cases when the cecum has been explored these fecal masses are so adherent to the walls that when removed there is sometimes bleeding of the mucous membrane beneath. This must of necessity cause a low grade of inflammation of the cecum which will frequently extend to the appendix. In such cases, on operation when a chronically inflamed appendix is found, one must remember the primary cause is not in the appendix, but in the cecum, and the removal of the appendix will not be of benefit to the patient.

Wilms has described a condition affecting the cecum known as mobile cecum, which is a movable atonic cecum. But little attention is now attached to this condition, for here, too, change in position plays but a very significant role; it is the adherent fixed cecum which is the seat of stasis and which is productive of symptoms.

By means of the Roentgen ray and screen we are able to determine the presence of adhesions surrounding the cecum, the position of such adhesions and how far these interfere with the motility of the bowel. Important advantages are frequently obtained by means of the fluoroscopic method in the study of the cecum and its surrounding structures. By means of palpation under the fluorescent screen many important conditions may be noted. It is by this method of examination that the identification of various areas of pain on pressure, the question of motility, separation of loops of intestines surrounding the cecum may be observed.

4. *Adhesions and Angulations.*—Attention has already been called to the angulations and kinks which may occur to the lower

right quadrant due to adhesions and which are usually readily recognized by means of the Roentgen ray. The adhesions may not, however, be limited to the appendix, but may be associated with the cecum, at times causing marked fixation of this portion of the intestine, or may be connected with the ascending colon or the ileum causing various degrees of obstruction, or may be due to pelvic inflammatory conditions producing a similar effect. In fact, the terminal portion of the ileum is a common seat for adhesions. On operation one frequently finds in cases of chronic appendicitis that the appendix itself is perfectly free and no adhesions are observed around the cecum, but the terminal ileum appears fixed and frequently kinked, so that the ileum beneath the kink becomes dilated and at times almost obstructed.

All of these conditions can be easily detected by the delay in the passage of the bismuth, which will be revealed in the plates, and by means of the fluoroscope.

Inasmuch as adhesions are detected by changes and delays in motility, great care must be exercised in drawing conclusions regarding these findings.

5. *Tuberculous Ulcerations.*—Brown and Sampson call attention to the importance of the Roentgen ray in the diagnosis of intestinal tuberculosis. According to these authors the clinical picture of intestinal tuberculosis is of little aid in the diagnosis in the early stages of this disease. On the other hand, the Roentgen ray may give definite information of colonic ulcerations, though the absence of the shadows does not exclude this condition absolutely. The Roentgen ray picture presents hypermotility of the bowel, complete evacuation often occurring in from 20 to 24 hours, though a small amount of barium may be left in the rectum at the end of this time. In many instances Brown and Sampson found the barium in the sigmoid, the rest of the intestine being empty, although a small gastric retention of bismuth was still present. In many cases the cecum and ascending colon manifested the greatest hypermotility. A most important sign is the spastic condition of the bowel, involving usually the cecum and cecocolon; the bowel has also an irregular appearance, presenting definite filling defects at the seat of the lesions. The presence of this intestinal hypermotility, spasm and filling defects give, according to Brown and Sampson, in a patient with pulmonary tuberculosis almost definite evidence of colonic tuberculosis.

While the above signs are associated with colonic tuberculosis, at the same time a very spastic irritable bowel will frequently be very improperly filled, due to spasm, and give almost an identical picture as that seen in tuberculosis, so that one must be extremely careful not to assume that every case of spasm and improper filling is due to tuberculosis.

6. *Ulcerations Due to Carcinoma.*—In this condition there is a definite filling defect in the cecum, due to the carcinoma. This defect is large, serrated and constant in all the plates. In addition, on fluoroscopic examination there is localized tenderness and fixation. Carcinoma of the cecum may exist for a long period of time before obstructive symptoms are noted. It is important in all instances to exercise great care to be positive of the Roentgen ray findings by both fluoroscopic examinations and bismuth enemata.

In a special instance which has come to our attention in which a capable Roentgenologist made a diagnosis of carcinoma of the ascending colon, and in which the diagnosis was confirmed by all the methods usually practiced by Roentgenologists, the filling defect and partial obstruction was found due to the dragging down of the bowel by adhesions connected with an inguinal hernia.

Diagnosis of carcinoma from filling defects should never be made from a single examination. If the bowel is not completely cleared the bismuth may flow around a small mass of fecal material, which will give a filling defect. In such instances it is always well to wait several days and then have the bowels thoroughly cleansed by means of cathartics and enemata and repeat the examination. If the filling defect persists in the same position, it is extremely unlikely that this is due to a bit of fecal material, but must be a true filling defect which points probably to malignancy. Small malignant masses, however, can exist in which the growth is so situated that even under the most careful examination no filling defects can be made out by means of the bismuth examination. The bismuth enema is the method par excellence for examination of colonic growths. A large-sized growth not producing obstruction may be frequently overlooked by means of the bismuth meal, but will almost invariably be shown by the enema method. This is particularly true when located in the descending colon and sigmoid, which are seldom properly filled when the bismuth is given by mouth.

In conclusion, we hope that we have shown the great aid afforded by the Roentgen ray examination in the diagnosis of lesions in the lower right quadrant. We must bear in mind, however, that this is but one of the many methods by which conclusions may be drawn concerning these intricate disturbances. Like all other methods of diagnosis, at times by faulty interpretation of shadows we may be led to a wrong conclusion. As with other lesions, the lower right quadrant disturbances should be studied in conjunction with the clinical signs. If the Roentgen ray interpretation is diametrically opposed to all the clinical findings and the two methods cannot be harmonized, it is probably wise to adhere to the clinical interpretation. In other words, no one method of diagnosis must be looked upon as absolute.

DISCUSSION.

DR. LEWIS GREGORY COLE, New York City: The facts that have been described in this paper are those which all of us have tried to observe.

In regard to the question of the ileo-cecal valve, I am sorry that I cannot be here to discuss the papers this afternoon, when we shall have some light thrown on that question, but I have to be in New York. I believe, however, that it is a more important factor than we have been led by Dr. Baetjer and Dr. Friedenwald to believe by their casual passing over the subject. I will grant that there are some cases in which a small amount of barium seeps through without any more pressure than is used in filling the colon, but in those cases in which it goes through and up, not only one or two feet, but up to the jejunum or even to the duodenum, I cannot help but feel that that condition is not a normal one.

I think that the cases should be divided into three groups—those in which a little of the barium seeps through, those in which mechanical deformities of the valve are observed (in which one can see that the valve is deformed), and those in which a large amount of solution passes through.

I have seen this condition of the ileo-cecal valve. I observed it and wrote an article in reference to it, but I laid this article aside for six years before publishing it and then I published it in its original form. I wrote two articles which I laid aside and then published later in their original form.

I think that the co-operation referred to in the paper is the thing we all need. There is no question about it. Antagonism gets us nowhere. Neither do competitive methods of diagnosis add to co-operation. It is a great temptation to set up a case and compare it with certain autopsy findings. It leads, however, to lack of co-operation and it is this lack of co-operation that we want to get away from.

We want to have the co-operation referred to in the paper, and I believe it is coming rapidly.

Dr. Jacob Kaufman, New York City: I think that the plea for closer co-operation between the roentgenologist and the internist or surgeon is a very excellent one; and the statement that the physician should not demand too much of the roentgenologist is very correct, too. After all, the examination by the X-ray is only one method of examination; and one should not ask for the impossible. If the X-ray finding fits in with the other findings and the history of the case, it is very valuable; but if not, its value as evidence should very carefully be weighed.

Dr. Baetjer mentioned carcinoma of the intestine, and cases in which a positive diagnosis was made by the X-ray and no carcinoma found. Much more disagreeable than the positive X-ray finding in cases where there is no carcinoma is the negative X-ray finding in cases where there is carcinoma. I bring up this point because I have quite a list of cases in which, on account of the history, carcinoma was suspected, but in which not one, but several, Roentgen-ray examinations gave negative results. Experiences have taught me not to be influenced in such cases by the negative X-ray findings. When my suspicion of cancer was sufficiently corroborated by one or another symptom, even if a positive diagnosis could not be made, I had these patients opened up at an early date, and in quite a number of cases carcinoma was found. The roentgenological examination fails too often in the examination of this type of disease of the gut, particularly when there is no marked obstruction.

I should like to know what Dr. Baetjer's experience is on this point. It is important for the practitioner to know, when he has a suspicion that he is dealing with a carcinoma, what course he ought to take. Only a short while ago, I observed such a case in which I based my tentative diagnosis of carcinoma on the abrupt onset of digestive disturbances in a man who, up to that time, had been practically well. The X-ray examination and that of the stomach contents and feces had proved perfectly negative, but in spite of carefully conducted treatment there was continued loss of weight and little improvement of the digestive disturbances. As the general picture was that of abdominal carcinoma, I disregarded the negative X-ray and laboratory findings. At operation the growth was found in the upper part of the jejunum, a very rare site; about three feet away from the beginning of the jejunum. I have had similar experiences with cases of cancer of the colon.

The visible palpable stiffening of the gut is, to me, the most reliable sign of obstruction.

It frequently happens that patients with obstructing cancer of the colon get symptoms of obstruction, especially sudden attacks of severe pains, only at long intervals. I have seen cases where the intervals lasted several months and even a year. When you examine such cases during an attack (and often even between attacks) you frequently find the stiffening of the gut as convincing evidence of the obstruction. And yet I have seen cases, in which this reliable clinical sign of obstruction had been noted, while the X-ray examination gave no evidence of obstruction.

Dr. Seymour Basch, New York City: I think that this is one of the most important chapters in clinical medicine, especially clinical medicine associated with roentgenology. Problems of the lower right iliac fossa are every-day occurrences, and it is only through association of the various departments of medicine and surgery that we can reach a more correct definition and interpretation of these problems. The X-ray is a part of this study, and must be associated with the laboratory findings, the clinical history and, if necessary, the results of surgical procedure, in order to give us better knowledge and data for future determination. One of the important functions of the X-ray, as Dr. Mills brought out in his paper and as Dr. Baetjer stated, is the localization of the organ in which the disease occurs. Thus Dr. Baetjer has mentioned a stomach that was displaced down to the iliac fossa.

The question of the visualization of the appendix is a difficult one to answer, and one that is not by any means settled. Some roentgenologists go so far as to state that it is only the appendix that does not visualize that is pathological. Others state that if they find an appendix visualized, they do not report anything but that it is fixed or not fixed, curled or not curled, and retains its contents for so many hours. I would weigh these statements only in connection with the history and the other findings. The most important point, to my mind, is that we can use the X-ray to differentiate conditions in the region of the appendix that otherwise we could not determine. Many patients complain of pain in the right iliac fossa, are operated on, and have their appendix taken out; and yet, after a short time, the symptoms return. In such cases, the X-ray would have shown that it was a dilated or motile cecum, an ileocecal incompetency, adhesions or angulations, etc., that lay at the bottom of the trouble. In some cases, tumors, particularly benign tumors, are present. Thus I had a case of a mucocele of the cecum. The man complained of just this type of symptoms. One could feel a mass, but could not determine to what organ it belonged. The X-ray showed that it was in the cecum, and operation verified the diagnosis.

Again I have patients who were operated on and, presumably, had their appendix taken out, and yet the X-ray later showed that the appendix was left in. I had a patient in Brooklyn who had undergone an operation and been told that his appendix had been taken out; he had an X-ray examination made later showing the appendix curled up in the right iliac fossa. In a large majority of cases of marked incompetence of the ileocecal valve and (I agree with the authors that the slight ones can be disregarded, provided that the symptoms do not point to the right iliac fossa and can be explained on other bases) associated neuro-muscular conditions will explain the trouble. The ileocecal incompetence is merely a part of that association.

Dr. Franklin W. White, Boston, Mass.: I was much interested to note that in speaking of the appendix, Dr. Baetjer laid very little stress on its position. The appendix slides around so in different patients and in different positions of the patient, that it is extraordinary how differently it looks at different times. The matter of tenderness, however, I had supposed was really of considerable value, if the tenderness over the appendix were contrasted with the tenderness near by. If contrasted with other signs, I should think that it would be of considerable value.

I should like to hear what Dr. Baetjer's idea is about the visualization of the appendix. If the appendix does not fill, is it retrocecal, or is something wrong with it?

I am also interested in incompetence of the ileocecal valve, and should like to know whether Dr. Baetjer knows of any normal case in which this test has been tried, filling the large intestines very full. My impression is that perhaps one-third or more of the persons who consider themselves normal would be found to have incompetence of that valve.

I may say, in passing, that we very much regret that Dr. Lawrason Brown, of Saranac Lake, will not be here this afternoon. He is not able to come, on account of his own illness. If Dr. Baetjer has had any experience with intestinal tuberculosis in the ileocecal region, he might say a word or two in regard to it.

Dr. I. Harris Levy, Syracuse, N. Y.: In this discussion there is one thing that has been forgotten, and that is the diagnosis of calculus in the ureter. I have seen cases in which the appendix was removed and the symptoms went on as before, and a calculus in the ureter was later discovered. Every suspected case of chronic appendicitis should be studied roentgenologically and also cystoscoped before operation is done.

Dr. Baetjer (closing): In reply to Dr. Cole, I would say that in the case he spoke of as an example of a mistake in diagnosis was an obstructed (?) case. It was tight. The stuff was just massed up there, and a gastro-intestinal examination was asked for.

Regarding ileocecal incompetency, I feel that if we can bring about a condition of incompetency without it being pathological.

In reply to Dr. White, I would say that a great many of these incompetencies were observed in cases in which there were no symptoms pointing to the lower right quadrant condition. I make bismuth enemata examination almost as a routine. If the enema bag is elevated too high, the valve under pressure of the enema yields, we get incompetency. When the bismuth is given by mouth, we find at times incompetency. Of how much value that is, I cannot say. I thought that Dr. Kaufman, in taking the attitude that he did towards roentgenology, was very wrong. He said that if the X-ray finding is contrary to clinical data, one should throw it out. He also spoke of a case in which X-ray examination showed that the condition was not carcinoma, and yet he had the patient explored. He threw the X-ray finding out because it did not agree with the rest of the examinations. I think that he was doing just what he objected to.

Regarding the ureteral stones in the region of the appendix. All of us who do much work have had such cases come up. I saw one case of an appendix that had dilated into a cyst and was filled with bismuth.

Regarding some of the questions that Dr. White asked: the question of filling or non-filling of the appendix and its position, and whether it is pathological when it fills or does not fill, and when you can or cannot move it about, I would say that I think that it is undoubtedly pathological when it does not fill. I do not see why a perfectly normal appendix should not fill. When the appendix has gone on to an obliterative type, you cannot fill it with bismuth and it is in a pathological state. I think that it is a little bit unwise to draw too arbitrary a conclusion from the filling or non-filling of the appendix.

In reference to tenderness, it may be that my technique has not been perfect. It seems to me that it is rather difficult to localize absolutely the tenderness, particularly when you have only a part of the appendix filled, and that is next to the cecum. If you have a long appendix filled and it is tender over the tip, you can draw conclusions. When the patients improve and you get them back and palpate over the place where the appendix is attached to the cecum, you find the same tenderness as before the operation. Pressure on the abdominal wall over the appendix region might be on the cecum, and we must therefore make a certain degree of reservation in drawing conclusions from tenderness. I am just giving you my own experience. Of course, we may say that it may be due to faulty methods of doing it and faulty interpretation.

In regard to the question of incompetency of the ileocecal valve, I would say that in normal patients, by elevating our enema bag, we have succeeded in getting incompetency in a good percentage of cases.

Dr. Jerome M. Lynch, New York City: Does Dr. Baetjer mean to state that by using a certain amount of force, he can overcome the effect of the ileocecal sphincter, and that ileocecal insufficiency is not pathological? I always had the impression that the force necessary to overcome the ileocecal sphincter would be sufficient to rupture the bowel provided the sphincteric tone was normal.

I would like Dr. Baetjer to state his position regarding this matter.

Dr. Baetjer: There comes up in my mind the question, What is normal? I think that when we give an enema, we bring about a distinctly abnormal condition of the large bowel, but not a pathological condition exactly. We fill up the entire bowel and cecum completely. When we fill the cecum, we find it more or less distended. Are we not probably mechanically influencing the ileocecal valve? I have never seen it under the fluoroscope and recognized it as such. I think that we bring about an abnormal condition by the use of the enema. I would not say that there is anything wrong with it, however. Some patients will have symptoms from making a routine examination. We have made the bismuth go into the ilium. In people with no symptoms, I have obtained this incompetency.

DR. LYNCH (questioning Dr. Baetjer): May I ask what the patient was examined for, if there were no symptoms? Patients with this condition do not have symptoms referable to the right iliac fossa. They are usually referred elsewhere. If the gut is filled on the left side, you will find that the distress is in the right. They have pain at other than the point of impaction.

DR. BAETJER (further answering Dr. Lynch): We have a certain class of cases that come to the hospital for study and have a routine X-ray examination made whether there is anything pointing to conditions down there or not. For instance, one was a cardiac case, other examinations were made for patients with no gastric symptoms at all, and yet by the enema method with pressure incompetency was obtained, which was not present by the bismuth by mouth examination.

DR. FRANK SMITHIES, Chicago, Ill.: We found that if we elevated a two-liter bismuth enema can more than 26 inches we could "force" the ileocecal valve nearly every time. We did this at the time when enthusiastic surgeons were re-making ileocecal valves to remedy so-called "incompetence." It is probable that the Roentgen enema material is tolerated in the colon and the cecum under protest. The endeavor is to send it back toward the anus. During that period of activity, there are recurrent cycles of physiologic incompetence of the ileocecal valve. If the cecum happens to be filled when the valve is open, there is, of course, a backflow into the terminal ileum; and if one happens to take a Roentgen plate at that time, one gets "incompetence" of the ileocecal valve.

A CONSIDERATION OF THE GASTRIC TEST MEAL FROM EXPERIMENTAL DATA.

By Drs. Martin E. Rehfuss and Philip B. Hawk,
PHILADELPHIA.

Gastric analysis resolves itself into an analysis of gastric function or functional analysis and etiologic or specific analysis. These two conceptions must be sharply differentiated, before we rationally approach this subject. The time has now come for us to definitely define the meaning of gastric analysis. This demands that before we lay down the foundation stones for the interpretation of gastric analysis, that we have an exact conception of the nature and limitations of the gastric response in health. The literature is full of the results of gastric analyses in widely diversified clinical conditions. But the important point and the one around which the results of all such analyses must revolve is the knowledge of just what exactly constitutes normal and pathological gastric function. For instance, we read that hyperacidity occurs in so many cases of gall-bladder cases, so many cases of chronic appendicitis, so many ulcer cases and yet no one has clearly defined exactly what constitutes hyperacidity. In our laboratories with the collaboration of a number of chemists, we were able to demonstrate that 42 per cent. of 842 complete gastric curves with all varieties of foods gave an acidity of 100 or over. Furthermore, we were able to demonstrate that while the average total acidity for beef and beef products was 120, for eggs the average total acidity was 80, and for vegetables it was 70. In other words, we were able to demonstrate clinical hyperacidity as estimated by titration findings as an absolutely normal phenomenon with some 40 per cent. of normal men and animal proteins give persistently high acidities easily classified from clinical standards as equivalent to those encountered pathologically. These contributions occurring in the *American Journal of Physiology* emphasize the one important fact that we have failed to base our clinical interpretations on the knowledge of normal limitations in acidity. The conclusion was forced on us that there were several normal types, one clearly belonging to this group and individuals who do belong to this group show characteristically with every variety of foodstuff an hypersecretory response. Another conclusion was that there are normal, fast and slow stomachs just as there are morphologically steer horn and J stomachs as normal variations under the X-ray. Furthermore, it is equally clear that the gastric response varies with the type of the meal and there is more or less specificity in the response to various foods.

Again in the consideration of low acidities, it is clear that with the exception of certain foods, this phenomenon is not a normal one and from our clinical studies it is equally apparent that the analyses of low acid curves reveal a variety of mechanisms, all of them resulting in somewhat similar curves. For instance, constitutional disturbances and severe anemias can give rise to the same sort of low acid curve that the entire gastritis group can give, but the former is accompanied by simply a depression in secretion and the latter not only by secretory depression but the tangible evidence of mucosal inflammation. Again there is unquestionably a form purely nervous and finally there is a form in which the elaboration of secretion by the mucosa is intact, but in which the low acid curve is due to excessive regurgitation of alkaline duodenal secretions. In other words, it is evident that widely different mechanisms can bring about similar conditions in the secretory output of the stomach.

I propose, therefore, in this paper to briefly review the subject of gastric analysis, as I see it, based on the contributions from our laboratories and attempt to outline a crude but sufficiently satisfactory system for the interpretation of gastric analysis. Before, therefore, we discuss the question of the analysis of pathological curves, I would call attention to the following important facts which form a basis for the consideration of pathological data. In other words, we must have a normal standard and a normal standard demands not only the average responses but also the limitations encountered in health.

1. Gastric digestion is a series of recurring cycles in which digestive phases alternate with interdigestive phases (fasting or resting stomach). Both of these phases must be investigated in an analysis of gastric function.

2. The normal fasting stomach shows a continued secretion averaging from our studies about 50 c.c. with an average total acidity of 30 and a free acidity of 18. I would place as the outside

limit of total acidity 70 and the outside limit of the free acidity as 50, although these figures, or rather limitations, may have to be altered. There should be no gross food retention, no pus or blood, and only traces of gastric mucus. Bile is present in over 50 per cent. of cases and trypsin in practically all, indicating a tendency toward regurgitation during this period.

3. The digestive phase in health varies with the type of the individual, whether hyper-iso or hypersecretory, all of them being adequately described in contributions on this subject and secondly according to the nature of the meal. In normal individuals, 74 per cent. of the beef experiments showed an acidity of 100 or more, while only 22 per cent. of the egg cases, 44 per cent. of the responses on milk and cream, 19.3 per cent. of the vegetables and 7.3 per cent. of the bread and cereals gave an acidity of 100 or over. If, therefore, the average acidity of beef was 120 and vegetables only 70, it is safe to assume that a series of figures which represented the test meal responses for beef must be very different from those obtained on eggs and those on milk, and finally those obtained with toast and tea.

4. The gastric curve is clearly made up of psychic and chemical secretions, both definable by separate laboratory procedures. Today we can accurately measure the psychic secretion, the chemical secretion, the continued secretion in the fasting stomach, and the amount of duodenal regurgitation.

5. Hypersecretion is a phenomenon seen in about 40 per cent. of normal individuals.

Therefore it follows that with the above facts clearly established, we are ready to examine the stomach from several different angles:

1. A test meal to demonstrate the maximum acid output—this is obviously most readily accomplished by the administration of a meal of 100 grammes of beef which, in health, shows an average total acidity of 120 and an average evacuation time of two hours and 35 minutes in the rapid emptying time, and three hours and 25 minutes in the slow emptying time.

2. Motor function may be demonstrated as above or far more satisfactorily by means of the simple Ewald meal with limitations given below.

3. To demonstrate pathological products from the gastric wall

the water meal proposed by Austin is a gastric stimulant and has the advantage of showing any abnormality in gastric output.

4. The psychic secretion is obtained for sight, taste and chewing of food by introducing the fractional tube and performing these several steps preferably with 100 grammes of chopped meat, allowing the subject with the tube *in situ* to see, smell and taste the cooking beef.

5. A pure chemical curve may be obtained by injecting the meal through the tube.

6. Test meals to reveal specific factors such as soluble albumin are those in which this substance is reduced to a minimum, as in the Ewald meal.

For the purposes of ordinary analysis, however, we clearly want to get information from two separate standpoints, one as to gastric function, the other as to the specific evidence of gastric disease, namely, the demonstration of histological, chemical or bacteriological evidence of disease.

For the purpose of functional analysis, which is not specific analysis and must not under any consideration be construed as such, we require a fixed standard and an exact knowledge of normal function. We will never be able to correlate gastric findings with a diversity of test meals. While meats, fish and milk give the highest acid values, breads, cereals, fruit and then vegetables give the lowest normal values. Only six cases out of 82 experiments on bread and cereals gave an acidity of 100 or over, indicating that the average output for bread is comparatively low, but emphasizing nevertheless that HIGH ACID FIGURES OCCUR IN HEALTH and that high acid figures are less important from a pathological standpoint than low acid figures. But one thing is clear, high acidity is of far more significance with bread and cereals from a pathological standpoint than it is with meats and animal proteins.

The simple Ewald meal of 25 grammes of toast and 240 grammes water or tea appeals to me as the simplest meal and has the double advantage of inducing a moderate acidity with rapid evacuation time. We can, therefore, accurately delimit:

1. *The Evacuation Time or the Measure of Motor Function.*—An Ewald meal in health is evacuated in from two to two and one-half hours. Anything at one hour and one-half or less or more than three hours, can be considered outside the normal

boundary lines. In health we encounter slow and rapid stomachs, but only rarely do we encounter figures in excess or below these limitations. I would, therefore, from a motor standpoint, divide all pathological responses into two groups (barring a third pathological group giving normal evacuation time) one with too rapid evacuation time or hypermotility and the other with too slow evacuation time or hypermotility. From a study of my clinical cases, I have found that hypermotility can be ascribed to four causes: (1) a group of subacidity and achylia cases; (2) a very small percentage of duodenal ulcers; (3) a form of scirrhous carcinoma converting the pylorus and antrum into a funnel; (4) a group due simply to peristaltic overaction including those very motile stomachs which the French have called "gastric chorea."

From the standpoint of delayed evacuation or hypomotility, we have both intra and extragastric causes, the slight delays being found most frequently with gastric myasthenias, atonic stomachs, and muscle defects beginning dilatation, while the marked delays are due to intragastric causes such as contracting pyloric ulcer, carcinoma of the pylorus, hypertrophic pylorus, benign tumors in that region, pylorospasm, gastric syphilis of the stenosing type, contracting ulcer of the lesser curvature with organic biloculation and, as extragastric causes, adhesions to the pylorus and duodenum, tumors interfering with gastric evacuation such as tumors of the head of the pancreas and so forth.

From a *Secretory Standpoint,* with this meal we have every variation, from exaggerated secretion or hyperacidity and hypersecretion to subacidity, anacidity, achylia and delayed gastric acidity. In practically every case the variations in the secretory output can be placed in four categories: (1) nervous, (2) circulatory, (3) mucosal, (4) a disturbance of the tryptic regurgitant mechanism.

I prefer, therefore, in the analysis of the secretory curve to divide all gastric secretory curves into four groups, depending on whether or not they are associated with definite evidence of diseased gastric states. For purposes of analysis one might divide all gastric responses in disease into four groups:

1. Clean digestion, simple secretory variations.
2. Secretory variations plus gastric mucus=indicating inflammation of the mucous membrane.

3. Secretory variations plus blood (intragastric)=indicating ulceration of the mucous membrane.

4. Secretory variations plus blood, pus, mucus, bacteria indicating ulceration and infection of the mucous membrane.

Group 1. *Clean Digestion.*—A clean digestion, and by that term we mean no evidence of mucosal irritation or disease, whether there be functional alterations or not, indicates in the presence of gastric symptoms THAT CONDITIONS OUTSIDE THE STOMACH ARE RESPONSIBLE FOR THE GASTRIC CONDITION AND THAT THE LATTER IS USUALLY A SECONDARY MANIFESTATION. For instance, a larval hyperacidity may be encountered in psychic disturbances, or a digestive or post-digestive hyperacidity or hyperacidity may be encountered, in which case the ultimate analysis will usually reveal factors outside the stomach inducing secretory changes. It is this clean group which is so frequently encountered in certain biliary disturbances, appendiceal disease, abdominal adhesions, and so forth. The assumption is that most frequently the effect is produced over nervous pathways and less frequently through the circulation. Clean subacidities and achylias are encountered in metabolic conditions, such as gout, thyroid disease, pellagra and almost any toxic condition which inhibits the vagus or stimulates the sympathetic, or through the circulation produces direct inhibition of the gastric mucosa. This is particularly true of those types associated with focal infections and anemias.

Group 2.—A secretory variation with the addition of a pathological element immediately introduces the element of gastric pathology. The commonest pathological element is gastric mucus, which must be differentiated from swallowed mucus. The definite and persistent presence of this element immediately introduces us to a very large group—the gastritis group. There are many varieties, in fact, some time ago I was able to collect over 200 cases of chronic gastritis induced in a variety of ways, direct irritants representing the whole line of dietary indiscretions, and food infections, the great group associated with irritants carried by the blood stream and a third group in which the chronic inflammation was secondary to diseased conditions occurring in most of the body viscera. By widely different mechanisms, these factors are able to produce similar lesions.

Group 3.—The additon to secretory variations of blood (meaning intragastric and not swallowed blood). Blood in the stomach means ulceration whether it be minute hemorrhagic erosions, acute ulceration, multiple erosions or chronic round ulcer, or whether it means ulceration or a neoplasm such as carcinoma or gumma. While it is true that a certain proportion of gastric ulcers do not bleed, nevertheless the continued presence of this element immediately indicates the element of ulceration. I have seen stomachs at operation which revealed blood and no visible spot of ulceration, nevertheless there was intense congestion. In our series of ulcers, only 38 per cent. showed high acidities and 30 per cent. comparatively low acidity. With ulcer of the duodenum an almost pathognomonic finding is blood with tryptic regurgitation or bile and only at those periods.

Group 4 is the group in which there is unquestioned evidence of INFECTION AND INFLAMMATION OF THE MUCOSA. In other words, the presence of blood, pus, mucus and bacteria indicates ulceration, inflammation and infection of the stomach, and I have only found this syndrome in three conditions true infected gastritis, gastric carcinoma and gastric syphilis. With the exception of rare forms of acute and phlegmonous gastritis, the presence of one of these three can be surmised if it can be demonstrated that these factors are intragastric and not extragastric or swallowed. The latter is discrete and does not persist throughout the curve, while the gastric material will be found throughout digestion and particularly toward the end of digestion.

The fact that alterations of the gastric secretion with persistent mucus content indicates gastritis by no means ends the investigation. As we have pointed out, an investigation must be made as to the correlated causes likely to induce such a disturbance. The analysis of the subacidity, anacidity, achylia and delayed digestion curves demand, therefore, a careful study of all the associated factors.

My idea is simply to propose a rational basis for the study of gastric findings. The establishment of functional data, such as the type of secretory curve and the evacuation time, does not make a diagnosis, but it constitutes evidence which, when correlated with other factors above mentioned and the general summary of the case, are of the greatest importance. Every gastric curve is a sum total of a series of factors which must be critically analyzed.

It is a mistake to put the cart before the horse and say that because so many cases of gastric ulcer reveal hyperacidity, an hyperacid curve is indicative of ulcer. Gastric ulcer can and does occur with a normal secretory and motor mechanism, and as far as we can demonstrate no functional variation, a point which is simply another link in the chain of evidence TO INDICATE THAT CERTAINLY SOME FORMS OF ULCER HAVE NEITHER SECRETORY OR MOTOR VARIATIONS AS ETIOLOGICAL FACTORS, BUT SOME OTHER CAUSE, PROBABLY TOXIC, TO EXPLAIN THEIR PRESENCE. Gastric cancer in its inception must exist with little or no demonstrable change in function. Gastritis induces changes in function and as such they are readily demonstrable and the whole line of muscular disturbances induces changes in evacuation and as such they are readily demonstrable. Let US REALIZE HERE AND NOW WITH GASTRIC ANALYSIS AS WITH THE X-RAY THAT NEITHER LIES BUT IT IS OUR INTERPRETATION WHICH IS FAULTY. We found out long ago that we could not make our analytical findings fit our preconceived notions of what the case ought to show. The attempt to trace the gastric responses in many of the diseased conditions is laudable, but more laudable is the attempt to analyze in a given case the factors which have brought about such a change.

In the present state of our knowledge the following facts seem justifiable:

1. Gastric analysis is either functional or specific and in both fields it has its limitations. The fractional analysis of a test meal is an exact determination of secretory and motor function, and the results of these two studies can demonstrate only alterations in function. Functional alterations may be functional or organic. To the analysis of motor and secretory function should be added the demonstration of specific pathologic elements such as blood, pus, mucus, bacteria, etc. Only the evidence of cancer cells or the persistent presence of pathogenic infecting organisms can be considered pathognomonic.

2. The interpretation of gastric analysis presupposes a knowledge of the normal gastric output. This is now well defined for both the periods of gastric activity and rest. These normal variations are given above. The secretory normal response can be divided into three types, but in disease any variation can occur.

DISEASE MAY ALTER EITHER THE DIGESTIVE OR INTERDIGESTIVE CURVE, OR BOTH, IT MAY ALTER FUNCTION AND IT MAY ADD ITS SPECIFIC ELEMENTS TO THE SUM TOTAL OF GASTRIC WORK.

3. There is no pathognomonic curve in any gastric condition. There is no pathognomonic curve in gastric cancer, ulcer, the inflammations, or, for that matter, any of the gastric conditions. We can only say that it may produce and usually does produce definite types of secretory and motor alterations and definite forms of pathological elements.

4. A sane view of this subject is urgently necessary and most of all is needed constructive work on the factors which induce alterations in function as well as the isolation and demonstration of those pathological elements which indicate true gastric pathology. Nowhere else is this so essential as in the study of gastric carcinoma.

5. We must have clearly defined the variations in health factors which we have attempted in our laboratories and already indicated on studies in beef, pork, lamb, milk, eggs, vegetables, and in articles shortly to appear, on the effect of the entire line of foodstuffs on normal gastric digestion.

6. This paper is presented in the hope that it will suggest a basis for the logical interpretation of gastric findings.

DISCUSSION.

(See page 237.)

ERRORS IN THE DIAGNOSIS AND TREATMENT OF DUODENAL ULCER.

By John Dudley Dunham, A. B., M. D.,

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Duodenal ulcer without complications is to be considered in this paper.

The anamnesis will always be considered the most important single factor upon which to predicate a diagnosis of this disease. After describing the typical history, Moynihan says: "It is therefore not necessary to the attaining of a diagnosis that any examination of the patient be made; the anamnesis is everything, the physical examination is relatively nothing."

In a footnote the distinguished author disclaims any desire to eliminate an examination in the diagnostic procedure. The history accepted as typical of duodenal ulcer is well known, but as has frequently been observed, a patient may present the classic history required in Moynihan's postulate without having the disease.

A diagnosis of duodenal ulcer should never be made without a thorough general examination. One requires chemical tests of the chyme, stools, urine, a blood count, a Wassermann test and, most important of all, fluoroscopy of the chest and abdomen. Lavage eight hours after a Riegel meal is also important, indicating at times a gastric stasis not shown with barium.

No one of the above measures will prove the existence of duodenal ulcer, but all will aid in the differentiation of other diseases with similar histories.

There are no chemical changes in the chyme typical of uncomplicated duodenal ulcer. Neither has the presence of occult blood in the chyme or stool any diagnostic value.

Hemorrhage is a late complication and proves neglect on the part of the clinician.

When we cease to recognize hyperchlorhydria and gastric neurosis as entities, we will overlook fewer early cases of duodenal ulcer.

Fluoroscopy of the chest will frequently disclose an enlarged aorta. This condition, with or without symptoms of an aortitis, is often present and suggests a syphilitic duodenal ulcer or visceral lues. Either of the latter diseases is easily confused with simple chronic duodenal ulcer.

Fluoroscopy of the pylorus and duodenum is an invaluable diagnostic aid when one sees an early hyperperistalsis followed by a later hypoperistalsis and duodenal distortion.

Bourcart* has found all patients with round ulcer or duodenum have sagging viscera.

This is quite contrary to twenty years' experience in my own work. Ulcer of the duodenum has rarely been associated in my patients with visceroptosis. In fact the presence of the latter has been considered a diagnostic sign against ulcer.

A Roentgen appearance of local constriction in the cap of the duodenum with local tenderness is strong evidence of duodenal ulcer.

Palpation for a tender spot should be made while the patient is in front of the screen. This is the only accurate means to associate the Roentgen defect with the pain point. In fact, pressure over the tender area will often cause spasmodic contraction which may be seen in the greater curvature.

Diagnostic acumen will not be improved so long as we are content to say the patient has either duodenal ulcer, appendicitis, cholecystitis or visceral lues. In most instances, painstaking study will enable one to decide upon the correct diagnosis.

Two patients with the so-called pathognomonic history were found to have an uncomplicated cholecystitis without disease in the duodenum. The following case illustrates another error in diagnosis after a rather careful history had been secured.

On January 26, 1914, Mrs. H. L., aged 30 years, was examined. Family history is negative. She had one miscarriage at three months' gestation. Five years ago she was operated upon for ectopic pregnancy. The appendix, one ovary and both tubes were removed. Six months ago she began to have burning, distress and eructations three or four hours after meals. The same symptoms are felt from 1 to 2 a. m. She is relieved by food or soda. The pain is never sufficiently acute to require opiates. There is occasional nausea. The appetite is good and a tendency to constipation exists. Neither vomiting, chills, fever nor jaundice has been experienced. The patient cannot eat acid foods or condiments without later distress.

Examination.—Hemoglobin, 80 per cent; erythrocytes, 4,100,000; leukocytes, 4,500. Blood pressure: systolic, 125 mm.; diastolic, 85 mm.

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*Rev. méd. de la Suisse romande, May, 1919.

Ewald test-meal.—Free hydrochloric acid, 64; total acidity, 92.

Benzidine test for occult blood markedly positive.

The stomach is empty six hours after a Riegal meal.

The stool indicates the presence of occult blood upon repeated examinations following a meat-free diet.

The urine is normal. Percussion, auscultation and fluoroscopy of chest are negative except for a slight enlargement of the aortic arch.

Fluoroscopy of the stomach reveals a persistent, rather extensive filling defect in the antrum pylori. Rectal and sigmoidoscopic examinations are negative. Diagnosis: Duodenal ulcer. An operation revealed a normal gall-bladder, pylorus and duodenum. The liver, so far as it could be observed, was covered with small yellowish-white spots.

The intra-abdominal glands were enlarged. A portion of the liver was excised, sectioned and found to show the histologic character of a gumma.

A few days after the operation a Wassermann test of the blood serum was two-plus positive, Craig system. Mercury and salvarsan effected an apparent cure, which has continued for six years.

Visceral syphilis is frequency accountable for errors in the diagnosis of duodenal ulcer.

The above cited case in 1914 persuaded me that a routine Wassermann test should be made upon every patient with chronic digestive disturbances. This practice has been followed for the past five years.

It is indeed surprising to note the frequency of lues as the cause of apparent duodenal ulcer.

In a review of my case records numerous instances are observed in which gastro-enterostomy failed to cure duodenal ulcer permanently. An unrecognized lues of the viscera unquestionably accounted for some failures.

Castex and Mathis* do not hesitate to affirm, on the basis of their personal observation, that before the age of 30 years, tardy inherited syphilis is the cause of 90 per cent. of the cases of gastric and duodenal ulcers and 10 per cent. are caused by acquired syphilis.

My own observation does not justify this high percentage. However, until the last five years some of my diagnoses have been incorrect.

After every method of examination—laboratory tests of chyme and stool and fluoroscopy of chest and abdomen have been em-

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*Abstract, *Jour. Am. Med. Assn.*, 1918, LXXI, 321.

ployed—one should make a thorough search for the stigmata of syphilis.

After making a diagnosis of duodenal ulcer, if one finds signs suggestive of syphilis the patient should be given the benefit of antiluetic treatment. This course is indicated even in the absence of a positive Wassermann test.

Internists, as a rule, will agree that the term "cure" following gastro-enterostomy is a misnomer and should be considered "arrested cases."

Late recurrence of duodenal ulcer is more frequently observed by the internist than by the surgeon.

Errors in diet with the use of condiments and acids will often cause a recurrence of the hunger and night pain, months or even years after operation. A patient is with difficulty convinced that life-long attention to proper diet is necessary to prevent a return of ulcer after gastro-enterostomy.

Occasionally operation fails to reveal a duodenal ulcer.

A patient with a typical duodenal history was operated upon and the surgeon failed to demonstrate any evidence of ulcer. A few days later death ensued, following cerebral hemorrhage. At necropsy a linear ulcer in the posterior aspect of the duodenum, with only slight cicatrization, was seen. This patient would probably have been amenable to medical treatment.

Rigid medical treatment in a hospital should be employed before a surgical operation is undertaken. I am firmly convinced that haphazard medical treatment is responsible for many failures.

Absolute gastric rest is impossible during life, but every effort should be directed to approximate that state.

The patient should be given an effective dose of oleum ricini with the beginning of the treatment. He should then be kept in a horizontal position in bed for ten to 14 days. During this time he must use the bed-pan and not be allowed to raise his head above the pillow. Thirst should be met with clysmata of water (not saline solution). Nutrient enemata may be added.

The duodenal tube is a foreign body near the seat of ulceration and should not be employed. It is, furthermore, usually a source of annoyance to the patient.

Alkaline drugs are well known as stimuli to gastric secretion at an interval after their ingestion, and should be omitted.

Nothing is to be given by the mouth during this ten to 14 day period. A modified milk and oatmeal diet is then administered at frequent intervals. Quantity and variety of non-irritating foods are then added during the remainder of the four weeks' hospital residence. At no time during the treatment is more than a three-hour interval allowed to elapse between feedings.

The patient must spend most of the four weeks in bed. Following the hospital treatment, six meals daily must be taken for at least one year. Irritating foods, such as acids, condiments and fried foods, are forbidden.

A regimen such as outlined above will arrest the symptoms and apparently cure an astonishingly large number of patients for long periods.

After the physician has convinced himself by antiluetic treatment or by rest cure in bed, or by both, that the patient is unimproved, then and then only should a surgical operation be undertaken.

DISCUSSION.

(See page 237.)

DETERMINATION OF THE NEED OF SURGERY IN PEPTIC ULCER, WITH REMARKS ON GASTRO-ENTEROSTOMY.

By W. A. Bastedo, M. D.,

NEW YORK.

A study of surgical statistics would convey the impression that in operative procedure we have a dependable cure for gastric and duodenal ulcer; and last year before this Association Alvarez said, "My own conviction after many years of seeing these ulcer patients returning with relapses is that our medical treatments do little if any good except in rare cases." Yet in our own practice, ulcer cases that have had one or more operations come for treatment almost as frequently as ulcer cases that have not been operated upon; and other gastro-enterologists tell us that this is also their experience. Such antagonistic ideas, therefore, furnish our excuse for this presentation on a somewhat hackneyed subject.

Ulcer cases may require emergency treatment or they may not, and this fact divides them into two groups.

1. *The Emergency Cases.*—The two striking emergencies which occur in gastric and duodenal ulcer are acute perforation and profuse hemorrhage. In acute perforation, all will agree, the earliest possible surgery is imperative.

In dangerous hemorrhage, on the other hand, it is quite largely the opinion that operative intervention is dangerous and mostly futile. Sir Berkeley Moynihan, in issuing a warning against hasty surgery, estimated that not more than 3 per cent. of profusely bleeding gastric and duodenal ulcers could be treated successfully by laparotomy. Lindberg of Copenhagen tabulated 68 cases of hematemesis so severe as to raise the question of an emergency operation. In all of the 68 surgery was decided against. Five died and at autopsy it was found that not one of these could have been helped by surgery. On comparing these statistics with others from clinics where operation had been the choice, Lindberg was convinced that surgical measures are never indicated in cases of acute hemorrhage from the stomach or duodenum. We find that many surgeons have had the trying experience of opening a stomach in such a case and were unable to find the site of the bleeding. Lund, of Boston, said, "I have learned that it is poor practice, when the patient is depleted by hemorrhage, to open the stomach and try to grasp the artery in the bottom of an ulcer." Other surgeons speak in like manner.

Yet now and then one hears of a case in which life has undoubtedly been saved by surgery, as where perforation accompanied the hemorrhage or where the bleeding point was readily found. We have in mind a recent case where a vein was held open by the indurated tissues, and where undoubtedly death would have ensued had operation not been performed. And it is a trying experience and one that seems inconsistent, for a physician to stand helplessly by while a patient vomits copious amounts of blood every hour or two and rapidly becomes exsanguinated under his very eyes. The difficulty lies in the fact that as these hemorrhages mostly cease spontaneously and are not fatal, we prefer to wait, and then when we decide that the bleeding probably will not stop it is too late for surgery.

In any case, therefore, while applying the proper medical measures, we should, where possible, prepare for transfusion. If the bleeding persists, transfusion is the measure of choice. If transfusion fails to stop the bleeding it at least puts the patient in better condition for surgery, which then in our opinion might be resorted to. Thus very few of the cases would come to surgery, but these few, being hopeless otherwise, might include a sufficient proportion of operable cases to justify the procedure. That is to say, the great risk of operative intervention and the small chance of helping by operation are overshadowed by the almost certainty of the patient's death if operation is not performed. In such a case the surgeon who fails is without blame.

II. *Cases Not in the Emergency Class.*—In the treatment of an ordinary ulcer case surgeons are not now so ready to operate as formerly, and many grant that judgment is required as to the need of surgery. For operation always produces a damaged abdomen and a damaged stomach with a new anatomy and a new physiology; it is followed only too often by undesirable sequelae; and frequently enough it leaves the patient relieved for a time, only to have him return later with a recurrence of symptoms which, because of the surgical damage, may be more difficult to treat than before. As Connell expresses it, "The immediate result is invariably most encouraging, but relapse is far from uncommon."

We cannot here discuss the merits of the various surgical procedures, nor compare one with another, but because it is the usual operation we shall dwell briefly on the subject of gastroenterostomy, referring you for much interesting data to the papers of Dr. Frank Smithies and Drs. Wilensky and Crohn, which have been read recently before this Association.

Gastroenterostomy.—"This operation," in the words of Frank Martin, "has been for years the cure-all for almost every stomach malady that exists." Of 261 ulcer cases returning for treatment after operation, Smithies found that gastroenterostomy had been done in all. But among the best surgeons today it would seem to be considered of value only for ulcers of the duodenum and of the pyloric and antral regions, and not, except in rare cases, for ulcers in the body of the stomach or high up on the lesser curvature. Indeed many eminent surgeons agree with Deaver that "when the ulcer is located elsewhere than at the pyloric end posterior gastroenterostomy does little if any good." However, Mills has directed attention to the fact that in certain hypotonic types of stomach, after a V-shaped or sleeve resection for ulcer of the lesser curvature a gastroenterostomy is required; we also have seen the necessity for gastroenterostomy in just such cases, and Peck has expressed the opinion that it is a wise procedure. Sir Berkeley Moynihan has said, "under no circumstances is the operation to be done in the absence of demonstrable organic disease." Yet this cannot be an absolute rule, for we have had good results in a few rebellious cases of marked gastric atony without ulcer.

Among the various reasons advanced in surgical writings to justify simple gastroenterostomy, those usually met with are the following: 1. That it relieves the pyloric end from muscular activity and from the irritation of food and gastric juice. 2. That it relieves the pyloric end from high peristaltic pressures. 3. That by being placed at the most dependent portion of the stomach it favors gravity drainage. 4. That it favors stomach emptying regardless of gravity drainage. 5. That by permitting the alkaline duodenal contents to flow back into the stomach it decreases gastric acidity. Let us consider these seriatim:

1. *That it relieves the pyloric end from muscular activity and*

from food and gastric juice. To emphasize the stress laid upon this factor I would quote Richard Lewisohn, who wrote as late as 1918: "Even at the present day many surgeons consider gastroenterostomy a safe procedure to keep food material away from the ulcer-bearing area." In the majority of cases the ulcer-bearing area is the antral, pyloric or duodenal region. Without giving the data in detail I might say that it has been shown repeatedly, both by laboratory experiments and by the Roentgen rays (see 4 below) that after a gastroenterostomy peristaltic waves occur in the pyloric end of the stomach the same as before the operation, that to a certain extent and sometimes in their entirety the food and gastric juice are swept past the new opening and take their normal course to the pylorus, and that vigorous churning takes place in this end of the stomach. In 262 post-operative cases Smithies found chymification after a mixed meal good or fair in 90 per cent. Therefore we may say that gastroenterostomy does not result in relief of the pyloric end from muscular activity or from the irritation of food and gastric juice, though it probably lessens the degree of these.

2. *That it relieves the pyloric end from high pressures.* It is at the antral and pyloric regions that normally intragastric pressure rises to the highest point. Moritz found the intragastric pressure at the cardiac end 6 to 8 cm. of water, and at the pyloric end 20 to 30 cm. with rhythmic rises to as high as 60 cm. Sick found the cardiac pressures 7 to 16 cm. and the pyloric pressures 25 to 42 cm., and von Pfungen, in a boy with a fistula in the mid-stomach, found the maximum pressure at the fistula 19 cm. and that at the pylorus 162 cm. In ulcer cases with hypermotility, the pyloric pressure is probably quite high, and in cases with hypermotility and pyloric obstruction, whether organic or spasmodic, the pyloric and antral pressures probably rise very high indeed. In pyloric obstruction without atony, Crohn and Wilensky have shown that the hunger contractions may take on a spasmodic character, with correspondingly high intragastric pressures, a condition abolished by operation. They found that except in these obstruction cases hunger contractions were usually absent. Therefore although it is agreed that in pyloric obstruction cases without atony gastroenterostomy results in lessened pressures on the ulcer area, yet in cases without obstruction this is by no

means so apparent. Certainly in Sick's case a mid-gastric fistula failed to relieve the pylorus from high pressure.

3. *That by being placed at the most dependent portion of the stomach it favors gravity drainage.* At operation we have repeatedly seen surgeons, imbued with this idea, strive to select the lowest point of the stomach. And as Martin remarks, they even place the patient in upright posture after operation to favor drainage. The fact is that the lower border of the stomach changes considerably during digestion, in the nearly empty stomach the pylorus becoming ordinarily the lowest or almost the lowest part. Moreover, as the stomach fills it rotates on an axis extending from the cardia to the pylorus. It can hardly be supposed then that what is the most dependent point at operation in a stomach rendered atonic by the anesthetic and in a patient lying on his back, will remain the most dependent point for any length of time during the digestive period after the stomach has regained its tone and the patient is upright.

As a matter of fact, however, in the ordinary alimentary tract food is not propelled by gravity but by differential pressures. Cannon and Blake showed in cats that after gastroenterostomy, even though the animal was held up so that the opening was in the most dependent portion, water placed in the stomach did not run by gravity through the stoma into the jejunum, but passed through only in response to the force of peristaltic waves. Therefore, in the ordinary case gastroenterostomy does not favor drainage by gravity. In the emptying of highly atonic stomachs in atonic abdomens with low intra-abdominal pressure, gravity may play a distinct rôle; yet in these cases unless there is pyloric stenosis the results of gastroenterostomy are not highly encouraging.

4. *That it favors stomach emptying regardless of gravity,* that is, by peristalsis. In regard to this, I find a great discrepancy of opinion. In the X-ray studies of Cannon and Blake on cats fed with fluid boiled starch, the food passed regularly through the pylorus and not through the new stoma, with two exceptions, one where the stoma was in the vestibule close to the pylorus, and the other where the stoma was large and in the anterior wall about halfway between the two ends of the stomach. Except for these two cases the pylorus was regularly the exit chosen even though it was much narrowed by a ligature. This was in animals with no ulcer to cause hyperperistalsis and abnormality

of pyloric closure. That both in man and laboratory animals the food mostly does pass the new opening to seek the natural outlet has been shown by others, *e. g.*, Tuffier, Delbet, Kelling, Guibe, Troell and Lewisohn; and Haertel, after the study of a large series of radiographs following gastroenterostomy in man, observes that though in most cases food passes through both the pylorus and the new stoma yet the peristaltic wave is unchanged and continues to be directed as before towards the pylorus. Schueller, Petrén, Cole, Carroll and other radiographers have made similar observations.

On the other hand, recent reports by C. H. Mayo, Carroll, Hartmann and Outland, Skinner and Clendenning point to the effectiveness of the gastroenterostomy stoma. And Carman and Balfour of the Mayo Clinic emphatically state that the opaque meal regularly passes freely through the stoma even after years. Several observers have remarked, however, that while in the first few weeks after operation the food has taken its exit for the most part through the new outlet, yet later it has returned in large measure to the outlet through the pylorus. This might be explained by assuming that in the first few weeks the ulcer is still in the irritative stage and is effective in producing pyloric obstruction by pylorospasm, but that later as the ulcer heals it no longer excites pylorospasm, and therefore as there is no longer obstruction, the food passes through the natural outlet. This would seem to be the desirable sequence of events. Truesdale showed that in some cases after the operation the pylorus, owing to disuse, undergoes atrophy and dilatation and remains permanently open. It is interesting to note how frequently in some of the relieved cases the X-ray shows a six-hour residue. Smithies found an eight-hour food residue after a mixed meal in 16.4 per cent. of those returning for treatment. Carter notes an early emptying time when both pylorus and stoma are patent, but delayed emptying when the pylorus is occluded at the operation. Wilensky and Crohn state that the emptying power of the stomach is not accelerated by gastroenterostomy with pyloric blocking, but is actually retarded.

5. *That whatever good is obtained is due to the diminution of acidity* by the passage of bile and pancreatic juice into the stomach through the new stoma. It is Deaver's opinion that if a posterior gastroenterostomy in ulcer of the pyloric region does

any good at all it is by allowing bile and pancreatic juice to enter the stomach, thus producing a neutralizing effect on the acid contents. This is the opinion of many surgeons. It is true that following gastroenterostomy the acidity tends to be reduced, for H. J. Paterson found that it falls on the average 30 points, and Smithies, in those returning for treatment, found a reduction in free acid and total acidity in about 80 per cent., the reduction averaging about 20 points, though in 17 per cent. there was no reduction and in three per cent. a rise in acidity. One month after operation Crohn and Wilensky found a slight average decrease in acidity though not a decrease in all cases.

The effect of bile and pancreatic juice on acidity.—Grey demonstrated by cholecystogastrostomy that when the total output of bile was made to pass through the stomach it did not materially affect the level of gastric acidity. He also deflected the total pancreatic juice into the stomach and likewise noted no material change in acidity. In a study of fractional curves after gastroenterostomy, Wilensky and Crohn observed that the influx of bile was frequently followed by a secondary rise in acidity and that at times the highest acidities were recorded when the bile was present in greatest abundance. These observations tally with our own experience and that of Smithies that highly bilious stomach contents after gastroenterostomy frequently show high acidities. H. S. Carter, Wilensky and Crohn have reported that a number of the operated cases gradually lowered their acidity till a complete achylia was developed. This could hardly have been due to a reflux of alkaline material. Therefore, though acidities tend to be lower after gastroenterostomy, they are not by any means always lower, and we do not believe that the diminished acidity is the effect of neutralization by regurgitated bile and pancreatic juice. As a matter of fact, the degree of gastric acidity is unimportant in deciding the improvement after operation (Troell, Crohn).

To summarize our remarks on the supposedly desirable effects of gastroenterostomy we might say that when there is an obstruction to the exit of food the operation permits stomach emptying and relieves the retention. But in *non-obstruction cases* it does not, at least to any great extent, relieve the ulcer-bearing region from peristaltic activity, from the irritation of food and gastric juice and from high peristaltic pressures, it does not favor gravity drainage, it does not as a rule accelerate the emptying time and it does not act through the neutralization of high acidity by the bile and pancreatic juice which it permits to enter the stomach. Therefore, except in cases of obstruction due either to disease or to operative procedure, as in pyloric stenosis or removal and possibly hour-glass contraction, we are unable to discover a satisfactory reason for a good result from gastroenterostomy. It would therefore seem a pertinent question to ask: Except in the presence of obstruction what useful function does gastroenterostomy perform?

Gastroenterostomy with pyloric blocking.—On account of the numerous failures of gastroenterostomy, and the frequency with which after gastroenterostomy the food has passed in the normal direction through the pyloric exit even when this was partially obstructed, a number of surgeons have attempted to block the pylorus by means of a silk ligature. W. J. Mayo has concluded that such blocking gives no advantage, and this belief is concurred in by Scudder, Gonnerud and others; and as it has not been a successful procedure it has largely been abandoned. But in its place has come pylorectomy or at least a severance of the duodenum from the pylorus. To this certain surgeons object on the ground that it makes the operation a more severe one, that it makes a permanent obstruction no longer needed when the ulcer heals, that it favors high pressures in the ulcer-bearing region, and possibly that by preventing the contact of hydrochloric acid with the duodenal mucous membrane it prevents the formation of secretion and hence results in a diminished secretion of pancreatic juice and bile. We do not think this last a factor, for three reasons, viz.: 1. The absence of hydrochloric acid in cases of gastric achylia is not accompanied by a decrease in the secretion of bile and pancreatic juice. 2. Hydrochloric acid can produce secretin in the jejunal mucous membrane, as shown by Bayliss and Starling. 3. In these cases there is obviously no lack of bile and pancreatic juice.

In several of our own cases after a simple gastroenterostomy had failed to relieve a secondary operation was done to block the pylorus. We can recall two in which the ulcer had been well up on the lesser curvature and had been excised by the saddle incision, but pylorospasm persisted. They did well after the pyloric excision. We can recall three others in which the ulcer was at or near the pylorus, and the pylorus at operation seemed well stenosed, but the symptoms continued after the gastro-enterostomy. All did well after pyloric obliteration.

Normal results of gastroenterostomy.—A gastroenterostomy regularly results in certain changes, viz.: 1. The passage of the acid and sometimes highly acid gastric contents into a jejunum which is habituated only to material that is alkaline. 2. Admixture of the gastric contents with the bile and pancreatic juice at an abnormally low point in the intestine. 3. The passage of some poorly disintegrated food into a jejunum accustomed to receiving material that is well chymified. After a mixed meal Smithies found that the gastric contents showed poor chymification in only 10 per cent. of the cases; yet it would seem that owing to the position of the stoma some of the food must be forced through without chymification.

Untoward sequelae of gastroenterostomy.—The most common untoward sequelae are: Renewed ulcer symptoms, retention symptoms, nausea and fullness without retention, pain and diarrhea.

Renewed ulcer symptoms after gastroenterostomy may result from:

1. Failure of the original ulcer to heal. After a remission of a few weeks or months brought about by the rest and diet following operation and similar to remissions after medical treatment the symptoms recur.

2. The development of a new gastric or duodenal ulcer.

3. The development of a true peptic ulcer in the jejunum below the stoma region.

4. The development of ulceration in or about the gastro-intestinal anastomosis. This is quite probably due in most instances either to trauma from the use of clamps as demonstrated by Troell, Coffey, White and Hamilton, or to the employment of unabsorbable sutures. Pool, W. J. Mayo, Mann, Gonnerud, Woolsey and others have reported finding the linen or silk sutures projecting from the bases of such ulcers. It may be due to careless feeding in the early post-operative period, for in a study of gastroenterostomy in animals, Flint found the new formed anastomosis to be the site of a healing ulcerated surface for about two weeks. These are the causes of renewed ulcer symptoms.

Retention symptoms may result from failure of the stoma or obstruction of the jejunum, because:

1. The opening is too far to the left.
2. The opening shrinks unduly. Finney had a case in which in spite of absolute pyloric stenosis the stoma had been reduced to the size of a straw. Von Eiselberg believes that reduction in size of the opening is almost always due to ulceration.
3. The opening is closed by distension of the stomach which causes stretching of the jejunum over the stoma. Cannon and Blake noted in cats that when the stomach was distended by a large meal the food passed through the stoma with difficulty, so they excised a gastroenterostomy stomach, tied its pylorus and filled it with water. As the gastric wall became stretched almost no water escaped through the stoma, the comparatively small jejunal wall having flattened out over the opening to become practically a part of the stomach wall.
4. The opening is closed by contraction against it of the cut circular fibers of the jejunum. In the words of Cannon and Blake, "The contraction of the cut circular muscles can have no other effect than to shorten and flatten the intestinal wall between the two lines of attachment." In two of our cases opened under local anesthesia vigorous peristaltic waves were present, and after the anastomosis had been made we observed that as each wave reached the cut in the jejunum the whole wall of the gut flattened against the opening, the effect of the contraction of the circular fibers that had been cut being just that described by Cannon and Blake. It was obvious that anything in the duodenum would shoot up into the stomach rather than pass into the distal limb of the jejunum.
5. The stoma or the jejunum is closed by volvulus or traction as when the stomach recovers from its operation atony or when the patient reassumes the upright position.
6. The food fails to pass readily into the distal limb of the jejunum, because of adhesions with kinking. The circular muscles proximal to this point being cut and stomach peristalsis being absent for many days following operation (Wilensky and Crohn), there is no driving peristaltic force to push the food along, break newly forming adhesions and straighten out a kink (Cannon). We have had two marked cases of obstruction by adhesions with pocketing of the jejunum at a distance of two or three inches

beyond the stoma, the X-ray showing immediate passage of food from the stomach, but partial obstruction at the lower point with pouching above.

7. The establishment of a vicious circle, the food that passes through the still open pylorus travelling back into the stomach through the stoma. Cannon and Blake watched such a process for half an hour, during which time not an atom of food passed into the distal limb of the jejunum. They noted no untoward results from the vicious circle. Vicious circle is usually due to a plastering of the jejunal wall against the opening or to obstruction of the distal limb of the jejunum. Probably less frequently than usually supposed is it a cause of untoward symptoms.

Nausea and fullness without retention may result from: 1. Too rapid emptying of the stomach. In feeding with the duodenal tube it has been found that passage of the food too rapidly or at a temperature much above or below that of the body is prone to result in feelings of distension, nausea, weakness or even collapse, symptoms quite the same as in occasional cases after gastroenterostomy. 2. Possibly, though not certainly, from too abundant regurgitation of bile. In practically all cases after gastroenterostomy bile is regurgitated into the stomach, yet no nausea ensues. But in two striking cases during the periods of nausea we have noted excessive quantities of bile in the stomach contents, while in the same patients when free from nausea there was very little bile. We have been inclined to attribute the nausea to the abundant bile in the stomach, though it is possible that the responsible factor may have been something else. For, though bile, when swallowed, is nauseating, yet ox bile introduced through a stomach tube is not so. (J. C. A. Gerster.)

Pain is usually the result of ulcer or adhesions, especially adhesions to the anterior abdominal wall.

Diarrhea is probably a sequel either to a too rapid emptying of the stomach, to irritation of the jejunum by either the acid stomach contents or coarse food particles, to changes in the pancreatic secretion, or to a superabundance of fat in the food. (Cameron.)

From Matthieu's Clinic at the St. Antoine, Paris, it was reported that very few if any of their cases of gastroenterostomy, if followed over a sufficiently long time, failed to show some pathologic symptoms, gastric or intestinal.

In a study of cases following posterior gastrojejunostomy with exclusion or removal of the pylorus, Wilensky and Crohn found that 11 did well after the operation, though it was necessary to curtail the amount of food permitted, 14 showed definite disturbances in physiology and seven showed mechanical disturbances in the functions of the gastro-jejunal stoma. In almost all there was absence of peristalsis one month after operation.

In the group that apparently did well motility and acidity were slightly diminished, and there was a distinct and persistent hypersecretion.

In the group with physiologic disturbances, there were mental depression with poor nutrition, pain, vomiting, occasionally hematemesis and constipation or rarely diarrhea. There were definite delay in motility, hypersecretion with a high acidity curve to the end of digestion, and a poorly functioning stoma, produced not by stomal closure but by disturbed muscular function.

In the group with mechanical or organic changes at the stoma there were markedly delayed motility with retention, a high acidity curve, violent peristalsis, six-hour Roentgen ray residue and insufficient or closed stoma.

Summary.

Considering all the possibilities we have mentioned, and other post-operative complications, such as hernia, rectus diastasis, gastro-intestinal fistula, and various unmentioned abdominal adhesions, and in addition the operative mortality and the number of cases that after a period of relief show relapse similar to that following medical relief, we must feel that surgery in an ulcer case must not be too lightly resorted to. As Wilensky and Crohn say, it is "evident that the impression that gastro-jejunostomy is an operation which in no way impairs the functional efficiency of the stomach is an erroneous one." "In only a minority of the cases does the stomach return to an almost normal state of functional activity."

We do not wish, however, to disparage the use of the operation in the proper case, and would say with Crohn that we could not treat disorders of the stomach without using gastroenterostomy. But its value is to make an exit for food when the normal exit is not available. And we hold with Torek that "the treatment of ulcer of the stomach is essentially medical and not surgical, and that it is only certain of its complications or sequelae that require surgical intervention." We now leave the subject of gastroenterostomy.

Cases of ulcer which require operation.—The emergency cases we have already considered. Of the others we have those in which surgery is imperative from the outset and those in which surgery should be resorted to only after thorough medical treatment.

Surgery imperative.—We consider surgery imperative and medical treatment futile in the following conditions, viz.: 1. Chronic penetration as shown by radiographs. 2. Palpable induration. 3. Adhesions which cause distortion of the stomach, interference with peristalsis, or much pain during the digestive period. 4. Permanent hour-glass. 5. Pyloric stenosis not syphilitic. 6. Repeated copious hemorrhages. 7. Conditions which suggest that an ulcer is becoming carcinomatous. In duodenal ulcer, as carcinomatous change rarely occurs, this is not of moment; but since in gastric ulcer this change is not infrequent, its possibility must induce an earlier consideration of surgery.

Surgery only after expert medical treatment.—The majority of peptic ulcer cases can, in our opinion, be definitely said to require surgery only after the failure of thorough and prolonged medical treatment. When a case is medical the relief of symptoms (not the cure) by treatment is, as a rule, quite prompt. Therefore, on the one hand, the failure of the treatment to relieve the symptoms suggests that the case is probably surgical; whereas, on the other hand, when a case seems in all likelihood surgical but not certainly so, a course of medical treatment is advisable to prove the point. Furthermore, if the patient shows a positive Wassermann reaction, or gives a history or any physical evidence of syphilis, antiluetic treatment should be tried.

Given a thorough medical trial by one competent to supervise the treatment, we should consider those cases surgical which continue to show, 1, persistent or recurrent hemorrhage even small in amount; 2, pain; 3, nausea; 4, pylorospasm of such persistence as to simulate pyloric stenosis; 5, inability to ingest comfortably the ordinary wholesome foods permitted by the circumstances of the patient, this making the poor patient a surgical case earlier than the well-to-do one; 6, inability to ingest comfortably enough food to maintain nutrition while living a normally occupied life, and 7, recurrence after apparently a cure. Or, as C. H. Mayo expresses it, "The best type of gastric case for surgery is that in which every method of medical treatment has failed, or the patients have become tired of the rigid diet and care which are necessary to relieve or to prevent relapse." This is our answer to the question when to operate in peptic ulcer cases. We shall not attempt to answer the question when to perform a second or a third operation if symptoms persist.

When an ulcer case is referred to the surgeon, the physician must still feel his responsibility to the patient, and in order to provide against having a surgical failure follow the medical failure should urge the serious attention of the surgeon to the following statements, viz.: That of Ginsburg that "small quantities of food at frequent intervals have a better physiological effect upon the recently short-circuited stomach than three daily meals"; that of Cannon that "after gastroenterostomy, effective action of the anastomosis demands that there shall not be more than a moderate distension of the stomach"; and that of Troell that "the recurrence of symptoms following operation in many cases depends to a considerable degree on the surgeon's neglecting to give dietetic and other prescriptions suitable for ulcer." We would commend the counsel of W. J. Mayo that "following surgical intervention the patient should be under good medical advice until permanent cure is assured"; and that of Carter that "after operation every opportunity for healing and cicatrization should be given, involving as this does weeks or months during all of which time the diet should be bland, soft and free from thermal, mechanical or chemical irritants." No longer should we tolerate a surgeon who places an ulcer case on heavy and bulky diet within two or three weeks of his operation.

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DISCUSSION OF PAPERS OF DRS. REHFUSS AND HAWK, DUNHAM AND BASTEDO.

Dr. John A. Lichty, Pittsburg, Pa.: When I was asked by the president to open the discussion of a paper by Dr. Rehfuss, I really felt quite concerned because I did not know just where I would find Dr. Rehfuss today in his work of investigation. Thus far his work has been rather revolutionary. He has thrown out most of our traditions and has smashed our idols and at times seems to have left us very little; but I think Dr. Rehfuss is coming to himself, as his paper evidences today.

It is said when the French physician Louis came back from Odessa to Paris, he resolved to spend seven years in the study of clinical medicine before he practiced. He was guided largely by the principle which he declared when he said: "The truth lies in the facts, and our mistakes come only from the wrong interpretation of those facts."

Dr. Rehfuss is trying to place before us the facts which underlie the physiology and the pathology of the gastro-intestinal tract. The methods he has suggested to us in the past few years are in themselves not absolutely perfect, but they have brought us on a long way in enabling us to get more closely to the facts. We are still, however, liable to misinterpret these facts and to draw wrong conclusions. The classification which Dr. Rehfuss has given us seems to me to be fundamental. It is very important to have a standardized test meal. This, of course, was insisted upon years ago by Prof. Ewald and every time we have strayed from the standard test meal we have been led into difficulties and contradictions. While Ewald has insisted upon the standard test meal, Dr. Rehfuss has shown us why the test meal must be standardized.

His classification of changes in the function of the stomach as manifested in the secretion, studied according to his method, seems to me is going to stand the test. He speaks of a clear gastric content of the normal stomach; the gastric content with mucus; the gastric content with blood; and that with pus and other tissues. The application of this principle in the study of chronic gastritis, I believe, should be quite helpful. I have for a long time felt that chronic gastritis, so diagnosed and interpreted, that is a condition associated with the symptoms of dyspepsia in which the gastric secretion is low and mucus is present, is really only a small part of a gastric disturbance which may have begun months or years before. We will assume that there is a certain time in a patient's history when the stomach is normal, the gastric secretion is normal, the motility is normal. That patient with indiscretion in diet or whatever the conditions may be which will bring about chronic gastritis, will begin to develop an oversecretion of hydrochloric acid. If he should be seen at that time, the diagnosis might very well be "hypersecretion of unknown origin" or due to indiscretion in diet. Later, mucus may appear, the secretion may be lowered and may even come back to the normal, only there are the symptoms of dyspepsia and in analysis of gastric contents considerable mucus is found. Later, probably months or years, the mucus is decidedly increased, the acidity is diminished, and probably here is the stage where the diagnosis of chronic gastritis is first made. Previous to this the concept of the case was always simply a functional disturbance. Any time during this previous history, if there is focal infection somewhere in the body or if there is traumatism to the stomach, ulceration may occur, no doubt, and probably later malignancy may arise.

Dr. Rehfuss' work brings out the point very definitely, it seems to me, but it is not one factor alone which brings about a diseased picture. Changes of gastric secretion may be one factor, the production of mucus another, changes of motility still another, infection and traumatism, others, and these together finally produce that pathology which we insist upon before a true diagnosis can be made.

Dr. Frank Smithies, Chicago, Ill.: Those papers that have been presented are all very good. They all teach something, certainly. Dr. Rehfuss' might be taken as a sort of follower for the one I read at New Orleans. In that paper I stated that it is wrong to regard gastric pathology as representing, usually, primary changes in the gastric mucosa, but that most of the gastric changes (with the possible exception of cancer) are probably secondary to things that have been initiated somewhere else in the body. We no longer, for example, regard gastric ulcer as a disease of the stomach itself, but as the local manifestation gastrically of an upset that may be dependent on a wide variety of causes. Consequently, any attempt to cure gastric ulcer must consist in a consideration of those factors associated with the production of the local gastric defect. If it does not, it becomes a process of flopping the patient from one doctor to another, until the surgeon gets him, does his durndest and then turns him back to the internist, and so on until someone makes an intelligent study of the individual and then attempts to treat the basic underlying condition.

In a series of gastric analyses, which I have printed elsewhere, I showed that of 1,728 proved peptic ulcers that we observed during the past nine years, the gastric acidity was no criterion as to the presence, the size or the degree of activity of the ulcer. This is to be expected. We can take the same patient and follow him through, and find that the pathology is either a progressive destructive affair or it is an attempt at repair. We find local, general and intragastric conditions entirely dependent on local and general pathology at the time of any single examination, and so different men may report different findings on the same patient at different seances, particularly regarding the facts of gastric analysis. In our 1,728 cases, the gastric acid and free acid followed closely on the figures which Dr. Rehfuss mentioned for his group. There were not quite 40 per cent. of meals showing so-called "hyperacidity"; rather more than 40 per cent. were what we called the normal range; and over 20 per cent. where there was "subacidity" or even free HCl. (These figures were printed eight years ago in *Amer. Jour. Med. Sc.*) Yet the disease went on clinically, just about the same in all these groups. Consequently, in treating peptic ulcer, we do not attempt to treat the acid factors as shown by test meals, fractional or single observation type, but we treat energetically the states which underlie ulcer production. We let the acid take care of itself, which it does. It is by instituting an ulcer regimen based on the above principles that we attribute whatever success we have had. Relief of symptoms does not mean cure; our efforts in treatment have been directed toward permanence of relief by actual cure.

With respect to gastro-jejunostomy, it affects ulcers, I think, because it alters the gastric physiology, secretory and motor. If you followed my work presented before this society some four years ago, you will observe that five years after operation practically all the patients had reached the pre-operative state with regard to the secretory factor. The acid reduction by gastro-jejunostomy was only temporary. The surgical procedure alters the peristaltic rhythm without permanently altering the secretory mechanism, except transiently as the result of various reflexes, stimuli or inhibitions.

These papers read have been all extremely valuable to me, particularly that of Dr. Rehfuss. His work points the way to those who are willing and able to learn, with respect to the treatment of these conditions. If we simply go on treating gastric pathology, particularly ulcer, from the side merely of intragastric chemistry, we are going to carry the acute ulcer, uncured, through to chronic ulcer; and of these, there are some which will go on to malignancy or cause other serious damage. If we realize the significance of and will treat the underlying conditions associated with peptic ulcer, we shall not have so many failures, nor shall we have the same patients passing from one doctor to another, and being treated for symptoms by a rule-of-thumb method over and over again by each and then failing to get well.

Dr. Bertram W. Sippy, Chicago, Ill.: There has been enough in these papers to make me talk about three minutes. The question is, what are we driving at in the treatment of ulcer? It must be clear that unless the ulcer is excised or resected, or is reduced in size by cautery and suture, it cannot fail to exist without going through a definite healing process, which consists in the development of granulation tissue, finally resulting in cicatrization of the ulcer. Now, manifestly, relatively few ulcers—in the past, at least—have been excised or have been resected. Relatively few are treated in this manner today. The surgical treatment of ulcer is gastroenterostomy, the world over; although more are being excised, resected or treated by cautery and suture now than formerly. This, however, was not done in the past.

Now all other ulcers must heal, if they cease to exist. Fundamental in the problem it should be that we should devote our attention to what is the cause of ulcer. What retards or prevents the healing of an ulcer, and what can we, as physicians and surgeons, do to promote the healing of ulcer? In other words, what can we do to promote the development of granulation tissue, which must finally lead to the cicatrization of the ulcer?

These are the problems that have been, and should be, before us. We cannot heal the ulcer. Nature fills in the granulation tissue. All that we can do is to remove those things that retard and prevent the healing. Until we know more about what prevents or retards the healing of ulcer, until we can get an exact knowledge of this, can we apply a rational treatment to the condition. I have been working on this problem a great many years. I have never said that I knew what prevents or retards the healing of ulcer. I think that no one knows. There may be many factors that are as yet unknown, but, taking what appear to be factors, we know that ulcers occur only where the gastric juice flows. That may be only a co-incidence; but 98 per cent. appear on the first part of the duodenum. Charles Mayo says that it is in this portion, where the acid chyme is retained until it is neutralized, that the duodenal ulcer occurs. Something in the gastric juice, therefore, prevents or retards the healing of ulcer. In our clinical and experimental work on the healing of ulcer by reducing a corrosion, lasting 24 hours to a twelve-hour corrosion by performing a gastroenterostomy. The effect of all treatment, when it is effectual in the healing of ulcer, would seem to be explained on the basis of a reduction in the duration of corrosion. All traumas heal readily. It has been noted for a number of years that experimental ulcers heal readily. It has been assumed that the raw surface of an ulcer produced by the injection of nitrate of silver has not lost its susceptibility to the gastric juice. This tissue, when brought together, will not have lost its resistance to the effect of gastric juice, and, therefore, healing takes place; but it is different in the case of the clinical ulcer, which does not heal. Why does it not heal? No one knows today; but it is assumed that the nutrition or blood supply is different from that of the experimental ulcer. The tissue has lost its resistance to the peptic action of the gastric juice, and, in proportion to its lost resistance, the gastric juice interferes with the healing of the ulcer.

Basing the treatment on the assumption that there are peptic ulcers, corrosive ulcers—certainly, the result has been to retard or prevent the healing; but basing the system of treatment on the proposition that there are digestive ulcers, peptic ulcers, corrosive ulcers and that gastric juice constitutes the hindrance, certain things happen. I will just briefly mention them. They have been published.

The effect of maintaining an accurate neutralization of free hydrochloric acid and thus completely annulling the effect of the gastric juice in cases of ulcer, has been noted. We have not less than 35 or 40 ulcers all the time under our management; and they are of the obstructive type, nearly all with peristaltic waves in the abdominal wall, and the serious kind that go to the surgeon. Eighty-five per cent. of all cases of pyloric obstruction will allow water or milk to go through in the seven-hour time. Fifteen per cent. will not. If we work on these later cases longer, half of them open up within seven to ten weeks; the rest require gastroenterostomy. The effect on pain is absolute. It disappears within a relatively short time and does not return. Hemorrhage is shown to gradually disappear under the influence of this management. I am talking of accurate management. If it does not stop, it is a suspected carcinoma or the diagnosis is wrong.

The penetrating type of ulcer gradually disappears. If it does not, you may be sure that it is not an active ulcer, but probably a carcinoma. There has never been a case that has perforated while under management. Excessive continued secretion is absolutely controlled. That is a problem in physiology. Gastroenterostomized cases that came with retention and excessive continued secretion were much improved. We have constantly in our service three to four such cases; and not long ago we had seven recurrences after gastroenterostomy. They go on and get well and remain well. We have large numbers of cases that have done so. Healing of ulcers takes place, from what can be learned by correspondence with cases, after years; and what is more, by operation afterwards. Thus, we have actually seen the development of granulation tissue in these ulcers. Surgeons say "that does not look like these ulcers; it has not the wide base." Autopsies on cases that have died of intercurrent diseases while under management have shown that the ulcers have healed. Some may be so deep and broad that they will never heal. What keeps the ulcer from healing? What can we do to promote its healing? Until we know more about it, we shall not have a rational treatment for ulcer.

Dr. J. C. Hemmeter, Baltimore, Md.: I hope that Dr. Rehfuss does not think that the lack of discussion on his paper indicates lack of interest on the part of the Association. The paper is full of information. It gave me an intellectual dyspepsia. I could not follow all the viewpoints that he brought out. I believe that he will make valuable contributions which will throw light on the physiology of the stomach.

I think that papers like Dr. Bastedo's are always necessary to clarify our views. We need the experience of men like that to strengthen us when we are in doubt as to the course that we should pursue. We have instinctively pursued this conservative course, but would like to be assured that non-operative-dietetic-mechanical methods of therapy have their spheres of usefulness.

Dr. Jacob Kaufmann, New York City: I would like very much to discuss the paper of Dr. Rehfuss, but it offers too much. I wish, however, to support him in his contention that we must differentiate various secretory types. It is important to know the type. What are high figures in one are normal figures in another type, and so on. I should like to say that the group of patients which have high secretory values are the ones that are inclined to develop ulcer. These same patients show other characteristic symptoms. They are high-strung individuals. They show a different type of blood picture; a definite type of blood-pressure valuations, etc. While we have done away with the conception that we can base a diagnosis of ulcer on the mere presence of high figures for acids, nevertheless I still maintain that the clinical picture of gastrosuccorrhea, as first described by Reichmann over 30 years ago, remains a valuable and reliable syndrome for the diagnosis of peptic ulcer. This clinical picture of Reichmann's disease is characterized by the finding of larger quantities of acid fluid in the fasting stomach of patients who regularly suffer with severe distress after meals, and particularly at night.

In treating ulcer patients, I think it is best to follow empirical experience, as Dr. Sippy stated, as long as we are not in a position to demonstrate more clearly the causative factors for the development of the ulcer. We must realize, however, the urgent need of studying the pathogenesis of peptic ulcer, so that we may be able not only to deal with an individual attack of the ulcerative process. The most characteristic feature of peptic ulcer is this, that it is a recurrent disease. One of the factors that has impressed itself on me is that these patients often go through periods of very great emotional and mental strain before a recurrent attack sets in. The period of mental and emotional strain has its effect on the action of the vegetative nervous system, causing motor and secretory irritation of the stomach, which brings on the formation of ulcer. Our study must be directed to examining the patient in every possible way in order to get the type of the patient, not only of his gastric secretion, to get his form of reaction, and then to find out whether by giving patients general hygienic treatment and by changing their mode of living we cannot prevent the recurrence of the ulcerative process.

DR. JULIUS FRIEDENWALD, Baltimore, Md.: Errors in diagnosis in peptic ulcer are not uncommon, inasmuch as the X-ray does not clear up the condition in a certain number of cases, due to the fact that the filling defect may not be revealed in the plates, or that we are dealing with complicated cases associated with adhesions.

In addition certain reflex and spastic conditions are at times mistaken for ulcer such as chronic appendicitis or spasms of the stomach, but these conditions can usually be excluded by careful examination.

The fact that ulcers are not detected at operation at times is well known. In a number of instances of my own this has occurred when a second operation revealed the true condition.

I agree with Dr. Dunham that surgery should not be resorted to in duodenal ulcers until after the patient has had a thorough medical cure, which has been found of no avail.

As a rule these cases do exceedingly well under medical treatment. I have employed the Sippy cure very largely during the past few years with exceptionally good results.

Dr. Bastedo raised the question of the effect of gastroenterostomy in peptic ulcer. At the meeting of this Association in 1915, Dr. Finney and I reported our experiences with gastroenterostomy comparing the results of 100 cases of this operation with a similar number of cases of pyloroplasty. We observed that the immediate effect of both procedures was quite good; but that the favorable result is not observed for some time, the stomach requiring two to three months before returning to normal function.

A normal motility was not secured until two or three months after operation. This was often delayed further by injudicious feeding. The end results which were obtained, that is as long as five years after operation, were much in favor of pyloroplasty cases; the percentage of satisfactory results being 88, while in gastroenterostomy it was 77.

Dr. J. P. Sawyer, Cleveland, Ohio: May I take a moment to call attention to one significance of test meals, which I think is a helpful point in understanding their use? Whenever any individual takes a meal into the stomach he supplies a physiological stimulus to that stomach. It is perfectly possible for digestion to be performed, with no acids present, over years; or perhaps with a high percentage of acid, no symptoms and no disturbances. When we give test meals, however, and remove stomach contents and examine them, so as to determine how that individual reacts at that particular time, and from the reaction at that time and repeated (as we have heard yesterday in connection with the X-ray examination, of the appearance of the gall-bladder and intestine) to judge of the condition of the patient, we must remember that it is only a continued reaction that furnishes a sound basis for inference. It is not on single test meals or single reactions that we are justified in basing a continued treatment. I feel sure that in looking upon the problem of digestion as a physiological process of great complexity, when we can thrash out our views finally, we have a logical foundation to base treatment on.

The psychic factor is often overlooked. It will at different times lead patients to react differently, at one time, with an achlorhydria; and in a succeeding meal, with a hyperchlorhydria. One man prominent in our profession was in my office one evening with severe distress with achlorhydria. He had a test meal the following morning, and I found a tremendous hyperchlorhydria. We find that repeatedly in numbers of cases. It was an instance of psychic stimulus, I am sure.

Dr. Rehfuss (closing): I did not expect to be pitch-forked into an ulcer discussion, when I came today. I did not say that hydrochloric acid in the gastric juice had anything to do with the formation of ulcer, but simply stated that there was no greater increase in an ulcer in gastric secretion than the incidence of high acid findings in normal cases.

We investigated some cases by Dr. Sippy's method. We were able to find that free hydrochloric acid was eliminated for that period.

Some years ago, I worked with Dr. Loeb on the production of acute ulcer of the stomach; and we found that we could produce it by introducing acid by means of the catheter and that we could inhibit it by keeping the stomach contents alkaline. There is no question that there is a secretory digestive nature to ulcer. I do not feel that any of us can say what the cause of ulcer is. I feel, however, that acid does not produce ulcer, but is probably the largest single factor in keeping up ulcer chronicity. That is not hedging at all.

The reason I discussed this subject is because there is an absolute need of some definite attitude on this subject. Certainly among the physicians that I come in contact with there is no definite principle or basis on which to establish any treatment. The gastric analysis seems to be here as good as waste paper. The gastric secretion goes through a regular cycle, and goes down to the resting secretion, which is altogether different. I do not believe that gastrosuccorrhea means ulcer. We had one normal individual with a continued secretion, which we followed 48 hours. The question is as to the quantity. There is a tobacco hypersecretion. I am speaking of gastrosuccorrhea and increased secretion, with a recurrent periodic painful condition, I believe, and this in my experience is very definitely associated with ulcer (I am going to close just what I have to say in a few words), I believe that the classification of gastric analysis is incomplete, and realize that in several years there will be an entirely different classification; but there is certainly a need for some new form of classification.

Dr. Dunham (closing): The confusion about gastric analyses and their results is clearly illustrated by an experience that I had some years ago. Professor Ewald called any acidity above 40 for free hydrochloric an abnormal secretion. When I said that the degrees of acidity were much higher in America, he replied, "In America, everything is colossal."

The object of my paper was to make a plea for an earlier diagnosis in gastric and duodenal ulcer. There is a beginning, and we must get at it. A few years ago, when a man made a diagnosis of gall stones, it was all right; but now, when a man allows his case to go on to the formation of stones, he is open to criticism. If you refrain from giving cathartics during treatment, you will have better effects by using soap suds enemas for evacuation.

Dr. Bastedo (closing): With regard to Dr. Deaver, I would say to Dr. Hemmeter that I am going to write Dr. Deaver about it. It was published in the *Annals of Surgery*, under a discussion in the public press some years ago, when we experimented with dogs who had a gastric fistula, in order to obtain gastric juice. We noted that when the laboratory man who took care of the dogs was cross when he came in the morning, and said to the dogs: "Get over there," when he cleaned their cages, we would not get any gastric juice; but when he patted the dogs on the back, and they wagged their tails, we got a copious flow. So I always say that it depends on how we wag our tails. I do not need to elaborate on that.

EPIGASTRIC HERNIA WITH SYMPTOMS OF PYLORIC OBSTRUCTION.

By Dr. Alexius McGlannan,

BALTIMORE.

Compared with the frequency of hernias of the lower abdominal wall, the epigastric form is an unusual lesion, making about one to two per cent. of the cases in any large series.

In my own small series there are three epigastric in a list of four hundred and seventy-eight hernias.

The etiology and development of an epigastric hernia is a matter of considerable interest. In one of my cases the protrusion followed a severe blow by a sharp-edged timber striking the upper portion of the left rectus muscle. Hotchkiss (*Annals of Surgery,* July, 1911, LIV, 78), quotes Roser as thinking "that the subperitoneal fat penetrates along the course of the vessels and nerves, often without the formation of a hernial sac; or in some cases the constant dragging upon this lipoma may result in sac formation and the appearance of a true hernia." Moschcowitz (*Surgery, Gynec. and Obst.,* April, 1914, XVIII, 504) proves Roser's theory to be the correct one.

Lathrop (*Boston Med. and Surg. Jour.,* February 25 and March 4, 1897) shows the frequent origin of epigastric hernia in the region of the round ligament of the liver. The rhombic outline of the apertures in the linea alba through which the vessels pass may make it easier for the protrusion of the subserous fat. (*Progressive Med.,* June, 1910, 46.)

That gastric symptoms may be produced by reflex action from peritoneal irritation is a generally accepted fact. Thus we may account for various digestive disturbances occurring in patients suffering from hernias of any kind or size. The epigastric form, however, may have a definite symptomatology, which in many ways resembles that of gastric and duodenal ulcer, except in the relation of the symptoms to the ingestion of food. Moschcowitz (*Annals of Surgery,* September, 1917, LXVI, 300), describes this symptom complex, and reports a number of corroborative cases.

There are periodic attacks of pain in the epigastrium. The patients complain of eructations and nausea, but rarely vomit. The ring in the fascia through which the protrusion occurs is always extremely tender. The area of tenderness is sharply defined and is superficial in situation.

Aaron (*J. A. M. A.*, May 13, 1916, LXVI, 534) describes similar symptoms as umbilical dyspepsia. Hall (*J. A. M. A.*, July 19, 1919, LXXIII, 171) describes an "electric bell reaction, a touch on the hernia causes instantaneous belching." This electric bell reaction was present in a marked degree in my traumatic epigastric hernia.

Hotchkiss (*l. c.*), gives the history of these hernias, the first of which were described by Arnauld of Villineuve in 1285.

Garangeot in 1743 recognized the hernia as the possible cause of certain obscure abdominal symptoms.

Terrier in 1885 insisted on the intimate relation of these hernias and various painful abdominal conditions.

D. D. Stewart (*American Medicine*, July 29, 1905, X, 185) reviews the literature on epigastric hernia, especially in its relation to gastric symptoms.

Gatewood (*J. A. M. A.*, January 8, 1916, LXVI, 85), could find no case on record in which the stomach made part of the contents of the hernia. The symptoms are always reflex in origin, unless they are due to actual disease of the stomach or duodenum.

The association of gastric or duodenal ulcer with epigastric hernia has occurred often enough to lead to speculation regarding the influence of the hernia on the production of the ulcer. While this relation of cause and effect is not clearly proven, the occurrence of both lesions in the same individual is frequent enough to justify the extension of the hernia operation to include an exploration of the stomach and duodenum through the wound. (*Prog. Med.*, June, 1911, p. 50.)

The two cases which I now report show the similarity of the symptoms of epigastric hernia with those of pyloric obstruction.

CASE 1. Surg. No. 14,383. This patient, a man 72 years of age, was studied through the courtesy of Dr. C. F. Blake at the Mercy Hospital, Baltimore. He had a large epigastric hernia which had been present several years. He came to the hospital on account of frequent vomiting, with loss of weight and strength. The study of the gastric contents showed absence of hydrochloric acid with food retention.

The X-ray examination indicated a pyloric obstruction, most likely due to carcinoma. An irregular contour of the greater curvature and pyloric region and hypermobility were noted with the fluoroscope. A filling defect and six-hour retention were observed on the plate.

At operation Dr. Blake proved the absence of any gastric lesion and the symptoms are therefore attributed to the hernia.

Case 2. Surg. No. 11,445. This patient, a man 62 years of age, had a right inguinal, as well as the epigastric hernia. He consulted his physician, Dr. Howard Jones, because of gastric distress with nausea, vomiting and epigastric distension, closely resembling the symptoms of pyloric obstruction. The diagnostic study made by Dr. Jones excluded a gastric lesion and pointed to the hernia as the cause of his symptoms.

On July 5, 1919, I operated on him at St. Agnes' Hospital, Baltimore, repairing both hernias, under procaine infiltration, and exploring the stomach through the epigastric wound. The absence of an organic obstruction was confirmed by the exploration. The epigastric hernia was a true hernia, having a thin sac and containing non-adherent omentum.

The patient has recovered from the operation and been relieved of all his digestive disturbances.

DISCUSSION.

Dr. Seymour Basch, New York City: This subject of epigastric hernia has always interested me, and I can substantiate what Dr. McGlannan has said. I cannot offer anything new, but believe I can say something of interest and importance on the subject. The literature is more abundant than we think. An excellent monograph was published by Kuttner in the early part of this century, and A. V. Moschcowitz also recently published one. Many of these cases appear masked under the ulcer syndrome, and usually with the diagnosis of gastric ulcer. There are two types of hernia, the visible and the palpable. The visible is the usual well-known epigastric hernia, but the other type, which appears without a tumor, is the dangerous type, because it is not recognized unless looked for. Major Hall of Camp Logan published an interesting paper on this subject in the *J. A. M. A.*, substantiating through a report and study of cases what we already know of the subject. It is only after operation that a cure is obtained.

I had a patient similar to Dr. McGlannan's first case, who had lost weight, had a complete achylia, and was suspected of having gastric cancer. He also had a palpable and visible hernia. The X-ray showed a large defect on the greater curvature on three different days. He was finally operated on with a negative result as far as the gastric tumor was concerned, but with a complete disappearance of the gastric defect on subsequent X-ray examination.

The hernia is not altogether, as Dr. McGlannan has said, a protrusion through a weak portion of the fascia, but as Dr. Moschcowitz points out, protrusion of preperitoneal fat alongside the blood vessels and nerves as they pass through the layers of the abdominal wall. I had a patient who was supposed to have an ulcer. Before operation I got permission to explore the abdomen, if necessary. We found a minute hernia at the intraabdominal wall; and on opening the abdomen found a chain of enlarged glands extending from the spleen to the hepatic flexure. The question was whether it was tuberculous, a lymphosarcoma, an abdominal Hodgkin's or another type of disease. Microscopical examination showed typical giant cells of tuberculosis. The boy recovered after operation. I saw him four years after the operation. He was perfectly well, and big and strong.

Another case was that of a doctor's wife, who was sent to me with a diagnosis of gastric ulcer. She had been operated on for appendicitis some years before. Her gastric symptoms were at first attributed to the appendix; but they were not relieved by the operation. She had a tender point in the median line without tumor. My diagnosis of epigastric hernia was not accepted by the doctor or by his brother, who was a surgeon. She went over the winter, and then she submitted to operation, a simple incision releasing the little hernia, and she has been perfectly well since. A third case was also referred to by Dr. Moschcowitz, who read his paper before the American Surgical Association two years ago.

One must not always look for a tumor. One must exclude ulcer, if possible, and then look for the tender point in the linea alba, etc. I have made mistakes in diagnoses in two cases: One with a persistent tender point in a patient who had suffered for ten years. He was operated on and it was found to be an ulcer. Another patient had a mass which proved to be a fat tumor that did not go through the wall; it was also a case of adherent appendix. He was operated on and recovered.

I do not think it is right to simply tell patients to wear a truss or support. This cannot cure the tumor, although it may give some temporary relief. The patient should be examined thoroughly and if we can exclude other conditions or cannot get benefit by treating other existent conditions, we should have the patient operated.

Dr. Charles G. Stockton, Buffalo: I rise to confirm by my own experience what has been said by the essayist and Dr. Basch, and to call attention to one fact which seems to me to show that my experience differs from the others as to the frequency of the occurrence of epigastric hernia. I do not think that it is a rare condition. It is a rare condition to find it with a conspicuous tumor, which can be readily palpated; but it is not rare to find, especially in the linea alba, where almost commonly it occurs, a defect that you can feel with the tip of the finger, a tender ring. By a study of the case, you find that the condition increases with effort on the part of the patient, that there is a slight impulse under the palpating finger when the patient makes increased abdominal tension, and that relief comes from operation. With Dr. Basch I find that any other treatment is disappointing.

In the past year I had an interesting case in a railroad worker, a trained man, who complained of symptoms of ulcer, and I believed it to be one of those small, comparatively invisible epigastric hernias. He suffered particularly when on his feet, and was relieved when in bed. I became convinced that it was not ulcer, and he was operated for hernia and went back to work, remaining well for some months. He had then to make an unexpected effort. He had to help in loading a prize hog into a car. One end of the box had been gotten into the car when the hog went back to the man's end of the box. The hog weighed three or four hundred pounds. The man made an effort to save the hog, and this effort caused a recurrence of the hernia. He was again operated on and cured, and is now well. I think that epigastric hernia in the linea alba is a comparatively common condition, and in making an examination I go over the linea alba inch by inch, to find whether there is any defect. The frequency with which this occurs and the relief afforded by operation have not been sufficiently brought to our notice.

Dr. John A. Lichty, Pittsburgh: In regard to the diagnosis, it is rather easy to mistake an epigastric hernia for a small lipoma of the epigastric wall. Several times this has come up when students were with me in a ward. In teaching I always insist that the student shall look for small lipomata in other parts of the body. These may frequently be found. There is also a peculiar feel in an epigastric hernia, caused by the omentum being pressed through the opening; it feels like wax paper. These two points should be considered before making a definite diagnosis of epigastric or ventral hernia. Why the symptoms of peptic ulcer and of epigastric hernia should be similar I cannot understand, and yet they seem to be frequently so.

Dr. Allen Jones, Buffalo, N. Y.: Sometimes the epigastric hernias are symptomless. A few weeks ago, there came to my service a man complaining of weakness, breathlessness, cough and slight edema. On examination, I found aortic regurgitation with a four plus Wassermann. I then examined the abdomen and discovered an epigastric tumor, which felt like a lipoma, but upon manipulation it was possible to reduce it and it proved to be an epigastric hernia. The ring was somewhat tender at its edges after the reduction of the hernia. On coughing, he produced the hernia again. He complained of no gastric symptoms other than those caused by the cardiac and old syphilitic conditions.

INFECTION OF THE DIGESTIVE TRACT: LATE RADIOGRAPHIC APPEARANCE OF DIGESTIVE TRACT IN CHRONIC ENTEROCOLITIS.

By Charles G. Stockton, M. D.,
and
Charles F. DeWitz, M. D.,
BUFFALO, N. Y.

Our knowledge of the bacteriology, anatomical state and physiological changes in infection of the digestive tract is too indefinite. That is the excuse for the presentation of this history of a somewhat unusual case. This case has been carefully studied and, with the exhibition of a few radiograms, I hope will be of interest.

A married woman of 36 showed signs of malnutrition, anemia and lassitude. There was hypochlorhydria, active gastric motility with no intestinal symptoms; the stools were well digested. After a period of mental rest, physical training and regulated meals followed by hydrochloric acid, the patient improved greatly and disappeared from observation for three years.

She then returned, suffering from diarrhea of several months' duration, unchecked by ordinary treatment. A gastric examination showed complete absence of secretion, the presence of mucus in excess, red and white blood cells, saprophytic bacteria and streptococci. The stools were liquid, acid and showed excess gas on fermentation. Cultures from the stools showed that the *bacillus aerogenes capsulatus* was the predominant organism.

A diet of lean meat and claret wine was selected, followed by temporary improvement; then ensued abdominal discomfort, alkaline putrefactive stools and the continuance of gas in excess. The diet was then limited to Bulgarian buttermilk. Hydrochloric acid was given, 15 drops six times a day, shortly after taking buttermilk; lavage was practiced daily. After cleansing the stomach it was rinsed with a solution of thymol one day, salicylic acid the next, and a weak silver solution the next. The patient made gradual improvement. The diarrhea was controlled, appetite improved, the patient gained in weight and returned to her home. There she resumed the ordinary diet and improved for two weeks, gaining 14 pounds. She then suddenly developed jaundice, but without fever or local tenderness.

Three weeks later she returned to Buffalo. The diarrhea had not reappeared; her appetite was fairly good; the anemia had not increased; there was a leucocyte count of 6,100, a hemoglobin of 78 per cent.; the temperature reached 99.6 in the evening. The stomach, which formerly emptied rather hurriedly, now retained the luncheon, consisting of solid food, for five hours. There had been no nausea. The gastric secretion had returned, total acidity being 20, free HCl 12 or .4 per cent., with absence of organic acids.

Physical examination showed a sligthly distended abdomen, hepatic dullness increased in area, the gall-bladder palpable with slight tenderness along the lower margin of the liver and over the duodenum. After washing out the stomach the duodenal tube was introduced. Through this was aspirated alkaline, somewhat turbid content, with bile and pancreatic secretion practically absent, containing much mucus, many pus cells and red blood cells. There was evidently duodenitis, closing the common duct at the papilla. When slight suction was applied to the tube blood appeared. The stools were formed, clay-colored. There was an unusual number of starch granules, meat fibers slightly changed, increase of mucus, many soap and fat crystals, no bile, no pus, no blood, free or occult. Cultures from the stools showed streptococci; on fermentation no gas developed. Cultures from the duodenal content showed many streptococci viridans.

Under treatment the duodenitis rapidly decreased; free bile and pancreatic secretion appeared in appreciable amount. A week later, although the stools showed bile color and diastatic ferment was present in the duodenum, there developed intermittent fever, with jaundice varying in intensity, characteristic of angiocholitis. In fact, although the duodenitis had subsided, the infection had extended to the bile ducts. After ten days the symptoms of cholangitis gradually subsided.

Treatment.—Duodenal feeding was established with milk and dextri-maltose, the end of the tube 32 inches from the teeth. Between feedings 1 c.c. of saturated solution of magnesium sulphate was slowly instilled through the tube, followed by a like amount of liquid petrolatum. Subsequently 0.50 c.c. of cold saturated aqueous solution of salicylic acid was used in the same way. After one week the tube was removed and a soft diet permitted; salicylic acid was continued. A few days after the beginning of the treatment, first the blood disappeared, then mucus and pus cells; the bacteria decreased in number, the bile and pancreatic ferments increased. After a few days of treatment bile reappeared in the stool. Palpation now showed an increase in the size of the gall-bladder. The obstruction at the papilla had decreased or disappeared, but there was now a mild cholangitis, perhaps cholecystitis, with characteristic recurrences of fever, as before stated.

During the remission there was subsidence of jaundice. The febrile recurrences became less intense, and with this there reappeared a marked enterocolitis, this time more severe than ever, with frequent large undigested stools, foul in odor, containing many gas bacilli, streptococci and colon bacilli. The color of the stools often changed, but they gave evidence of the presence of bile. At this time the stools became very large, with absence of all signs of pancreatic activity. There was much unchanged starch, much soap, many fat crystals, some free fat and undigested muscle fibers. It was decided that the infection had extended into the radicles of the duct of Wirsung, causing a degree of pancreatitis, corresponding with the cholangitis.

On two occasions there was moderate hemorrhage of comparatively bright blood in the stools. The fluoroscope and radiograms showed a dilated, inactive stomach, a moderate ptosis of stomach and intestines, with but little peristalsis, the colon being large, comparatively motionless, with incomplete haustra at long intervals with the bismuth meal, while on injection with the barium meal the colon showed practically no peristaltic waves, was dilated and gave the appearance of a large motionless tube, such as has been described as characteristic in colitis. There was some evidence of spasticity at the sigmoid. The sigmoidoscope examination made on several occasions revealed an edematous appearing mucosa, hyperemic, on one occasion showing marked congestion with enlarged follicles.

Improvement in the patient's condition and control of diarrhea began with the administration of small doses of opium and a "smooth diet" of mixed character. This time the Bulgarian milk failed to give relief.

The enterocolitis has largely subsided, but even yet there are occasional slight elevations of temperature for a day or two, always succeeded by an appreciable return of the icteric tint. Streptococci are yet found in the stools, also spores of the gas bacillus. There is general improvement in the patient's condition, and complete recovery seems probable. At present, while the patient is convalescent, there are intervals when the outflow of bile and pancreatic juice is undoubtedly diminished, and probably neither bile nor pancreatic juice has at any time returned in normal proportion.

LABORATORY NOTES ON THE CASE.

By Charles F. DeWitz, M. D.

Stools:

October 8, 1919. The *bacillus aerogenes capsulatus*, with spores, was for the first time discovered in the stool.

October 14, 1919. No free starch found. Stools heated, milk added, but no gas developed on fermentation.

October 23, 1919. Evidence of starch. Stools heated, milk added, gas present.

October 28, 1919. No free starch. Stools heated, milk added, gas one-fourth per cent. as indicated in the Einhorn fermentation tube. Gas bacilli found, also a few spores.

October 30, 1919. No gas in fermentation tube, no bacilli but many spores.

November 1, 1919. Neither gas bacilli nor spores. No gas on fermentation.

November 11, 1919. Same. At this time the diarrhea had disappeared; the patient went home, gaining in weight.

December 27, 1919. Patient returned with jaundice. Stools alcoholic. Very slight evidence of diastatic function in stools. No gas developed in starch solution on fermentation. Free starch present in stools, shown by microscope with iodine stain.

February 11, 1920. Moderate diarrhea. Welch's bacillus again found. Stools with starch solution made a slight showing of gas in Einhorn tube; starch unchanged (on fermentation). Fehling's solution not reduced. With dextri-maltose, gas developed to one-third per cent.

February 18, 1920. Mixture of stool with starch gave gas to one-half per cent. in Einhorn tube. No free starch remained and no reduction of Fehling's solution.

March 9, 1920. No free starch. Gas developed to one per cent. in Einhorn tube with dextri-maltose.

April 1, 1920. A few gas bacilli found; with boiled stool no gas developed with dextri-maltose.

April 6, 1920. Small amount of starch in stools, which now became very large, suggesting an inactive pancreas.

April 17, 1920. Large amount of starch in stools; no diastatic ferment. Stools very large, much fat present.

April 19, 1920. No starch seen in stools. Disappearance of gas bacillus.

Study of Duodenal Contents for Diastatic Function:

December 27, 1919: Duodenal content with fermentation shows only a trace of gas. (In normal duodenal content the starch is readily converted to sugar, and with further fermentation there is gas formation. No free starch is found. If fermentation is complete the content will reduce Fehling's solution.)

December 28, 1919. Small amount of gas developed from starch and duodenal contents in fermentation tube, with slight reduction of Fehling's. Starch converted to maltose or dextrose. Test made at time when the dextrins had not been changed to maltose or glucose, therefore Fehling's was reduced only to a slight extent. No free starch demonstrated.

December 31, 1919. No gas on fermentation. Faint reduction of Fehling's.

January 3, 1920. No gas on fermentation. Fehling's solution reduced 3+.

January 5, 1920. Gas on fermentation one-fourth per cent. in Einhorn tube. Fehling's solution not reduced. (Intermediate products of amylolysis do not reduce Fehling's.)

February 17, 1920. Starch solution mixed with duodenal contents showed on fermentation very little gas in 18 hours. Much unchanged starch. No reduction of Fehling's. Pancreas evidently inactive.

NOTES.—In the resorption process in the intestine it is interesting to note that the intestinal mucosa is only slightly permeable, not only to high molecular colloids, but also to disaccharids, but is permeable to the monosaccharids. The mucosa permits only those sugars to pass readily which can easily be used by the tissue cells, *i. e.*, the monosaccharids. Apparently the tissue cells are not adapted to dealing with the majority of disaccharids, as may be shown by the fact that if cane sugar or lactose be introduced parenterally, that is by cutaneous or intravenous injection, they are simply excreted without change. This is not true of maltose, because the blood contains the ferment "maltase," which is capable of splitting the parenterally introduced maltose into glucose after it has entered the circulation.

Observation shows that the influence of pancreatic diastase upon the starch from oats is such that it is quite readily converted into sugar. Observation also shows that potato starch, while very readily changed into achroodexterin, is only very slowly converted into sugar.

DISCUSSION.

DR. F. H. BAETJER, Baltimore: Dr. Stockton says that he is a little bit at sea in interpreting these plates, and has called on me to do it. I feel that it is a case of the blind leading the blind. I really do not know what to say about the case. In looking at the plate, I would disagree with Dr. Stockton about its being a sluggish, atonic stomach. He has a sharply contracted pyloric ring, which we do not usually see with a sluggish, atonic stomach. I feel that he probably did lay too much stress on the question of retention. When we talk of four and five-hour retention, we have to take into consideration the build of the individual. We have to divide the cases into the highly plethoric type, in which we have gravity with peristalsis; the typical cow horn type of stomach, in which we have gravity working a little bit against peristalsis; the fish-hook type, which is found in very thin persons with a scaphoid abdomen, in which you have gravity working against peristalsis, and, consequently, must have a longer emptying time. This looks like such a case. This is of little value, unless we know the type of stomach.

As to the question of the colon, we saw various spots of gas where the bismuth has not filled it out, and we get areas in the bismuth in which it is clear. I attribute that to flakes of mucus in the bismuth. Where Dr. Stockton spoke of the possibility of malignant growth, I think that the only kind of growth that could have produced it is an annular growth, and we rarely find the channel in the center of the bowels. In the second place, the edges are so clean cut, which one does not see ordinarily in malignancy. I should be inclined to believe that it was partly spasm and that the shreds going through were shreds of mucus incorporated in the bismuth. We have been giving an enema to patients and examining the bowel, and giving bismuth by mouth. The results have been disappointing. The enemata have shown practically nothing. We have not demonstrated any ulcerated areas. In some cases we got pictures similar to that of Dr. Stockton, and in others the opposite—namely, a very spastic colon. The only one factor in common seems to be a small amount of mucus with bismuth incorporated in it, which is not diagnostic, because we see it in so many conditions.

Dr. John A. Lichty, Pittsburgh: Will Dr. Stockton tell us whether the patient is gaining in weight now, and also whether there has ever been an approach to pernicious anemia in the blood picture?

Dr. Franklin W. White, Boston: It struck me that it was almost needless to make any remarks about the radiographic appearance, after Dr. Baetjer has spoken; but in looking at the picture of the stomach and bowel here, in the stomach, for example, the peristalsis was of moderate degree, but was certainly present in the lower part; and while the peristalsis of the colon was not marked at all, it certainly was present. In the enema, we got the round and more or less tubular appearance which is so common in an enema when a passive bowel is filled up under pressure. This picture we see very frequently, or, at least, one very close to it, in cases of congenital asthenia and ptosis, where the tone of the elementary canal is rather low all the way through. This condition was probably accentuated by the infection and further loss of time. The slow emptying was probably what we might expect in that type of case, and also the pressure on the duodenum. We get the biggest duodenal cap in people with no tone. A little, tired-looking woman will have a great big cap, while an asthenic individual will have a very small one. In chronic colitis we have a really tubular, more or less rigid colon, which fills up like a hose and empties like one, and really does not show any haustration at all.

DR. ALLEN JONES, Buffalo: The colon in these cases assumes the character of a sausage. I was impressed with the clinical evidence that we had of infection of the gall-bladder and of the pancreas. These are the features which are serious in the case. How far improvement will occur now, without drainage of the gall-bladder, is the question; and whether drainage, as suggested by Dr. Lyon, would cure is a question. It might be a good start.

DR. ANTHONY BASSLER, New York City: I think one shows an unwarranted courage in discussing another person's case, particularly when one has not had personal contact with the case and fails to sense the case in a clinical way. But it occurred to me, as Dr. Stockton described his case and described it so well, that it corresponded to rather a common type of disorder. Being in a woman of 36 years and of the type described, the fact that she had a chronic history of hypochlorhydria or achylia and a dilated stomach with some function, a dilated duodenum and a visible ampulla of Vater, suggesting pathology of the gall-bladder, a gas bacillus predominance, with an acid stool, a native protein diet producing an alkaline condition with putrefaction, and then a simple mixed diet producing a return of the gas bacillus and, particularly significant, the spores of the gas bacillus in the stools, the catarrhal picture and other points, suggested to me that perhaps he was dealing here with originally a mixed intestinal toxemia, in which the gas bacilli are merely secondary facultative organisms, and not important so much as the organisms of the colon group—that there had been after the infection of the intestinal contents, a submucous infection of the gut wall, and perhaps then what I have often met with, a granular degeneration of sympathetic nerves and plexuses of the abdomen accounting for the deficiency in motor function and the dilatation as well. Finally, there ensued an infection of the portal circulation of some kind, with a deficient bactericidal liver function, an infection then of the biliary canals, a cholangitis and cholecystitis.

Another point more on the diagnosis (which is the point at issue) is regarding taking bismuth by mouth and diagnosing spasticity of the colon. One often sees this when bismuth is given by mouth, whereas barium per rectum will show deficiency of peristalsis; and I think that this spasticity is the commonest finding in this type of intestinal toxemia, and the deficiency of contraction in the colon filled per rectum, while it is common, as Lyon has shown, is mostly significant of gut wall infection. The future treatment of this case is one of attention to the biology of intestinal digestion plus the patching up of such end results as have come on.

Finally, it is not uncommon, in studies at the operating table, at autopsy and in examination of the animals, to find that there are bacteria in the portal circulation; this fact is a subject that has not received as much attention as it should have received. As I pointed out to Dr. Lyon, when he was working on his method, and when he stated, as he did originally, that his fourth bottle of contents was sterile, he would find, if he made further study, that these last drippings from the liver were commonly infected. Since then, I understand, he has confirmed what I have been watching for some time. Your case, Doctor Stockton, occurs to me as an intestinal infection, submucosal, then portal, and finally, perhaps, biliary, and that many of the other sides of the case are secondary to this generalized abdominal infection.

Dr. Stockton (closing): I appreciate the discussion of the gentlemen who have been kind enough to contribute. Dr. Baetjer's decision on the question of atony of the stomach was very interesting to me. I think I shall have to learn a good deal about the interpretation of X-ray plates before I know all that there is to it; but from the clinical history of the case, there was no doubt about the atony. The patient, just before this X-ray work, had gastric stasis. That had occurred from time to time, rather corresponding with her improvement or depression.

Regarding achylia, she could not be said to have that. First she had hypochlorhydria, then she lost weight during the diarrhea and never regained it. All attempts to restore weight are balked by what I consider her pancreatic defect. The anemia is not marked and it never has been. It is secondary anemia. The hemoglobin, as I stated, was about 80 per cent., and her red count about corresponded. I suppose the blood is concentrated through the excessive watery evacuations. Probably she has a deficiency in blood mass.

In speaking about the colon, I have, of course, seen pictures of a good many instances of injected colons in all sorts of conditions, but have never seen a colon to correspond with this. It is not unlike, in its way of filling and emptying, a colon which has undergone fibroid change, only it is very large. With the fluoroscope, I think, you would say that it is different from the colon when relaxed or atonic. At least it so impressed me, and I think that is the impression that Dr. Jones received. Dr. Jones pointed out, at the time he saw the case, first, the probability of gall-bladder infection. I think it is probable that there was a cholangitis. I think she had had first, gastritis, then duodenitis, before we had any involvement of the gall-bladder or biliary tract. There was a time of definite lessening of the duodenitis, followed by an onset of cholangitis and with this the recurrence of bile and pancreatic secretion in the duodenal contents. Soon after there appeared the ordinary febrile attacks, the symptoms that go with cholangitis. The gall-bladder may have been involved, but probably not primarily.

The question has been raised of the probability of the infection of the mouth and upper air passages. That has been searched for. She has a good mouth. I never saw as clean a mouth with such an intestinal infection. I do not know where the infection came from. That she has some primary source of infection is undoubtedly true, but I have been unable to locate it.

I am much obliged to Dr. Bassler for his suggestions in regard to the case. I was much interested in what he said.

SOME FUNDAMENTAL PRINCIPLES UNDERLYING INTESTINAL AND RECTAL SURGERY.

By Jerome M. Lynch, M. D.,

NEW YORK.

Several recent studies have tended to indicate that human beings may be divided into groups or types according to their structural organization. Bean, who has studied racial types extensively in several parts of the world, has laid particular stress on the thin epithelial type and the robust connective tissue type. The first, or hyper-ontomorph, is a lank individual found to possess a long vertical stomach, short small intestine, and a long large intestine; the second, meso-ontomorph, is fat and broad in general appearance with a large transverse stomach and a small intestine and short large intestine. Each type, Bean believes, may be prone to a particular disease which occurs only rarely in the other type.

Stockard, studying from an entirely different standpoint, believes two chief types of individuals exist and that the one group differs from the other on account of having undergone its early post-natal development under a different complex of the internal secretions. The two types in the end correspond with Bean's—the hyper-ontomorph being an individual that has had a high physiological thyroid bring about early differentiation of organs and parts without excessive growth in size. The meso-ontomorph as a child had a low active thyroid, grew large in size, particularly in width, but its organ differentiation was slow. There are a number of correlated characters presented by individuals of characters shown by the other type.

Therefore a hyper-ontomorph, or high thyroid type, would be mentally precocious and physically frail. He would have a vertical stomach; the small bowel would be subject during early childhood to alimentary diseases. Certain ones of this type would have an acute costal angle; a long narrow chest; and a very active nervous system. On the contrary, a low thyroid type, or meso-ontomorph, would be physically well developed; have a transverse stomach; the small bowel would be very long and the large bowel very short. He would have a good digestion and metabolism. Between the two we have the hypo-ontomorph. The following history prepared by Professor Stockard is helpful in the classification of patients:

PERSONAL HISTORY.

Name.............................. Age.......... Sex..........
Date of Birth..............Place of Birth (in mts. or not).........
F., Nationality.............Place of Birth.......................
M., Nationality.............Place of Birth.......................
B..........................Place in Family......................
S. . ...Place in Family......................
Fam., Wt..........................Ht......................
Growth and Development after Puberty...........................
....
....
Sudden Changes in Wt. (10 lbs. or more)and dates................
....
....
Shape of Head and Face...
....
Interpupillary Distance............Vision: Hypermet.............
Size and Shape of Nose....................Hypomet.............
Prognathous Jaw.................Size of Lower Jaw.............
Larynx: Size......................Pitch of Voice................
Trunk (Length).................Extremities (Length)...........
Body Hair......................Age of Appearance.............
Skin: Texture.........................Pigmentation.............
Periodic Crying and Nervous Spells in Mother.....................
Idiosyncrasies........................ Digestion...............
Bowel Habit..
Urine Variation under Emotional Stress..........................
Physical Strength...................Endurance.................
Sex Function in Female................Menstruation: Age........
Onset......... Pain......... Duration......... Interval.........
Amt................... Temperament (Moods)....................
Pregnancies (Toxem., Labors, Breasts, Lactation).................
....
Sex Function in Male.......................Puberty............
Degree of Impulse...
Nervous Control.................Inhibitory Reflexes.............
....
....
....

The importance of this classification has a direct bearing on some of the surgical problems with which we are confronted.

The procedure so largely in vogue during the last ten years, of fixing organs in certain positions to make them conform to a conceived anatomical standard, is, in our opinion, a mistake in the light of our present knowledge of the great variation in size and position in different types. It is obvious that it would be incorrect to try to place the organs of a hyper-ontomorph in the position occupied by those of a meso-ontomorph. Now this is exactly what some surgeons have been trying to do by plication and other mechanical methods.

It is apparent that if a patient has a long narrow chest and an acute costal angle the most suitable position for his stomach is vertical, and any surgical alteration will be unfortunate. It is, therefore, fundamentally incorrect to try to convert this congenital type into something altogether different. This applies, with equal force, to the position of the colon. Plication, here, would be just as ineffectual as in the matter of the stomach. In fact, fixation of organs that should be movable is a surgical error. On the other hand, the fixation of movable organs that belong fixed, is surgically correct.

The cecum and sigmoid are two of the most variable organs in the body, and, when over-developed, are responsible for many digestive disturbances often referable to the stomach. A large movable cecum, using the apendix as a suspensory ligament, causes reflex stomach symptoms that might be mistaken for ulcer or chronic appendicitis. So, also with a long sigmoid. In the former case, the appendix may have to be removed, even though it is not diseased, and if an elbow deformity of the ascending colon accompanies a large non-fused cecum, with a mobility of 180 degrees, it may require a developmental reconstruction for its correction.

Another problem of importance in these operations, and particularly in removal of the colon, is the question of the action of the ileocecal valve and the behaviour of an intestinal stoma. Not all animals possess the ileocecal sphincter.

I am firmly convinced, in the absence of proof, that the various segments of the alimentary canal are capable of initiating peristalsis; and this is borne out by the perfect functioning of a new stoma after the removal of part of the colon. I am further convinced, if the necessity arose, as it might in the case of multiple adenomata of the colon, with beginning malignancy, that the colon could be removed and function restored by attaching the ileum to the anus.

It is my firm opinion that in cases of movable cecum and anchored appendix a low-grade infection starts in the appendix, and gradually ascends to the cecum and ascending colon, resulting in chronic appendicitis, perityphlitis, and colitis.

I have recently removed a colon of this kind, and the report of the pathological examination, by Professor Ewing is as follows: "Severe old chronic catarrhal enteritis and colitis. Hernias of cecal mucosa and wall. Marked chronic productive perityphlitis with adhesions and partial constrictions. Section shows chronic catarrhal colitis with extensive pigmentation of stroma cells."

Non-specific and specific purulent infections of the colon, continuous, discontinuous and segmental, are becoming increasingly common, and I feel that a great many of them originate in the ceco-appendix. I believe the colon bacillus is frequently the offending organism. We have had five cases in all where the colon bacillus was the only organism found. Three of these were relieved by vaccines alone; two required operation because of complicating strictures. It is interesting to note in connection with these cases that all complained of indigestion and all were quickly relieved by vaccines or operation.

The remarkable gain in weight, particularly after a stoma, brings up the interesting question of the influence of any irritation—whether infection or tumor—on digestion. The wide distribution of sympathetic impulse in contradistinction to the localized influence of the central nervous system, together with the loss of tone engendered by local irritation, may offer some explanation of the symptoms accompanying this disorder.

One of the fundamental surgical principles involved in the treatment of infections of the colon is that when an infection has passed beyond medical treatment it can be cured by placing the stoma cephalad to the infection. There is a widespread misconception as to the inconveniences caused by a stoma, and the number of operations that have been devised to overcome these so-called inconveniences shows that there is some misconception, or lack of understanding, of the principles involved. The simplest method is always the best. And in my experience the bringing out of a loop and suturing it in position is entirely sufficient, regardless of whether the stoma is a temporary or permanent expedient.

In not one of the cases in which we have performed this operation has the patient complained of any inconvenience, and very seldom if ever, do they use any mechanical appliance other than a piece of gauze to protect against accidents.

The sympathetic nerve supply to the bowel is more or less segmental, and it may be that each segment of the bowel has a potential sphincteric control; so that when a segment becomes terminal by operative procedure, a functional change gradually occurs and there is a slowing of the current as it approaches the end. Certain it is that some change takes place which permits the individual with a stoma to reassume control. In cancer, a stoma is of value only when used at the earliest possible moment after the tumor is discovered to be inoperable, and it is not of value when the patient is moribund.

Too often the surgeon delays till the bowel is obstructed and then such procedure merely hastens the end. The operation *per se* is one of the simplest in surgery, and except in fat people, can be performed under novocaine anesthesia.

One would think, from the multiplicity of methods that have been suggested and carried out, that an artificial anus functioned most unsatisfactorily; when, in reality, it functions perfectly. I frequently find that after an artificial anus has been made, a tumor, previously supposed to be inoperable, may be successfully removed on account of its diminution in size and the great improvement in the condition of the patient.

The small bowel is usually described as separated from the large by a valve—the ileocecal valve. Elliott has proved that this is not a valve but a sphincter muscle which contracts upon stimulation of the superior splanchnic nerves; and that its excitor cells are situated in the superior mesenteric ganglion. Langley and Anderson have demonstrated that the internal anal sphincter contracts on stimulation of the inferior splanchnic, and its motor nerves are situated in the inferior mesenteric ganglion.

The question then arises, what can surgery do to restore function in ileocecal insufficiency? The operation so far devised is conceived under a false impression of the anatomical mechanism and physiological function of this barrier between the small and large bowel. If the insufficiency is due, as it is in most cases, to a lack of sympathetic control, or perhaps suprarenal insufficiency, then no mechanical device known to surgery can avail. It is, therefore, conceivable that adhesions or an inflamed appendix, situated close to the ileocecal sphincter, interfering with its proper contraction, would cause a loss of tone. Under these circumstances surgical interference for the removal of the appendix or of adhesions would allow the muscle to functionate properly and return to a normal state.

This is the only condition where surgery in ileoccal insufficiency can be considered justifiable.

DISCUSSION.

DR. ALEXIUS MCGLANNAN, Baltimore: The ideal at which the surgeon aims in operating, is to remove the pathological process that is present, with the least discomfort to the patient. There are three main types. The first is amputation; for instance, of a limb. We have no way to restore a limb after amputation. Secondly, we have the group of operations in which, through the existence of a factor of safety, we can remove a certain amount of tissue without interfering with function, as in sub-total thyroidectomy for goitre. Then we have the third group in which, by adaptation, the function is carried on by the remaining portion of the body in a compensatory way. Resections of part of the gastro-intestinal tract comes under this class. In order that the function may be restored properly, a great deal of preliminary study and a great deal of new work must be done, if we are to plan our operation and maintain our ideal. The patient is studied, to determine certain characteristics of the gastro-intestinal tract and show what the surgeon hopes to accomplish by gastro-intestinal surgery. There is not a case of fixing organs that should be movable and making movable organs that should be fixed, which has not been discounted by the late effects. The patient does better when operated on for the acute lesion than in the case of secondary defects, where we have secondary disturbances to deal with. These complicate the picture and complicate the opportunities for repair. If the ileocecal valve is thrown out of function by the appendix, and the appendix is immediately removed, the restoration of function will be prompt; but if the operation is postponed until fibrosis has occurred, removal of the appendix will not restore function to the valve and the value of the operation is discounted. Dr. Lynch's research is work that will certainly add materially to the value of our gastro-intestinal operations.

ILEAL REGURGITATION, NERVES AND DIET, IN THE CHRONIC INTESTINAL INVALID.*

By John Bryant, M. D.,

BOSTON.

I. Introduction.

Since 1566,[1] it has been known that there is a structure called the ileocecal valve, and it has been known since 1570[2] that the valvular function of this structure is to prevent the regurgitation of feces into the small intestine. Furthermore, it has been known for 98 years[3] that the active muscular sphincteric function of the mechanism at the distal end of the ileum is to moderate the flow of the contents of the small intestine into the colon.

Actual incompetence of the ileocecal valve was first produced experimentally in 1586.[4] This condition has been recognized as a clinical entity for 23 years,[5] and its existence was demonstrated in the human by the bismuth-Roentgen method at least 18 years ago.[6] Yet Cole,[6] writing six years ago, was obliged to bemoan the fact that he was unable to arouse any interest in this subject, even on asking if symptoms might be expected, should a patient be fed colonic contents. Cole, Case, and other authorities in Roentgen work, have recognized a correlation in the degree of ileal regurgitation and the severity of the symptoms complained of by the patient, and the probable causative connection between ileal regurgitation and many cases of ileal stasis has frequently been pointed out.

Case,[7] in 1913, reported ileal regurgitation in over 16 per cent. of 1,500 consecutive gastro-enterological cases. Cole[8] believes that one might expect to find with modern technique, perhaps 30 per cent. of ileal regurgitation. Baetjer[9] has stated that it is possible to find as high as 50 per cent. of ileal regurgitation by including every case in which a little of the barium enema can be demonstrated to have entered the terminal ileum. Yet even today, almost no one can be found who will take more than the most casual interest in this subject in other than a purely academic manner.

If 30 per cent. of all patients coming for gastro-intestinal examination had regurgitation of food into the mouth all the time, someone would soon have to take interest. In the same way, if 30 per cent. of all gastro-intestinal patients suffered from chronic rectal incompetence, something would certainly get done about it. Yet just because the junction between the ileum and the colon is unable to cause external annoyance to the patient or his friends, nothing gets done about it. It is easy to create a furor about the appendix, or a gall stone, but not about the ileocecal mechanism. Why? Possibly because anatomists pass it over with a few lines of small type, medical schools pass it by in parenthesis, and students devote their time to something more obvious. Under these circumstances it is scarcely to be wondered at if the victim of ileal regurgitation turns in disgust from physicians who remain ignorant more than 400 years after its discovery, concerning an important anatomic and physiologic structure, in order to obtain perhaps a little alleviation of his symptoms at the hands of some cult not in the regular profession.

The term ileal regurgitation as here used includes not only regurgitation into the ileum resulting from incompetence of the actual ileocecal protective mechanism, but also the condition produced surgically by operative removal of the ileocecal area of the intestine; there is thus produced the same condition of continuity between the colon and small intestine. Two such surgically produced cases of regurgitation are included in the present study. For purposes of treatment, both these groups of cases are in the same class, on account of their lack of a break in continuity between the large and small intestine.

The objects of this paper are as follows:

1. To draw attention to the fact that too little attention is being devoted to ileal regurgitation, a pathologic condition which is a fact and not a theory.
2. To point out the ease and certainty with which the presence of this condition may be determined.
3. To present for consideration a new diagnostic sign of ileal regurgitation which is at least suggestive in value.
4. To indicate the remarkable frequency of ileal regurgitation in the class of cases referred to as chronic intestinal invalids.
5. To indicate the almost constant coexistence of ileal regurgitation and various forms of excessive nervous irritability.
6. To outline a non-surgical method of treatment which has given a reasonable degree of satisfaction.

7. To record the results of such treatment in a consecutive series of 50 chronic intestinal invalids.

II. DIAGNOSIS.

a) Hertz,[5] in 1897, reported a clinical sign, the presence of which was considered by him to indicate the existence of ileal regurgitation. Treatment carried out by him on the assumption of the existence of regurgitation apparently gave satisfactory results. His diagnostic procedure was as follows: One hand is pressed deep across the middle of the ascending colon to block possible distal expulsion of gas in the cecum. Downward pressure with the other hand in the direction of the pole of the cecum will then, if regurgitation exists, result in coarse crepitation and perhaps a gurgling noise as the cecal gas is forced back into the ileum.

b) Gas is not usually present in the small intestine. The presence of generalized gas throughout the abdomen suggests a deficient ileocecal mechanism.

c) A cecum and ascending colon dilated with gas, combined with a spastic descending colon, frequently accompany ileal regurgitation.

d) In the presence of this condition, the hepatic flexure and the sigmoid loop are frequently distended by gas.

e) A generalized yellowish discoloration of the skin, especially in the distribution areas affected by Addison's disease, is usual.

f) The eye sign is positive. By the eye sign is indicated a marked dirty discoloration of the whole of both eye sockets. This discoloration is not like other more or less limited discolorations about the eyes with which the writer is familiar. This sign is suggestive, not pathognomonic. It is practically always present, varying in degree with the intensity and duration of the ileal regurgitation. It may be present in the absence of such regurgitation. Should this be the case, however, treatment as for ileal regurgitation will cause a gradual disappearance of the sign in the same manner, but more rapidly than is the case when ileal regurgitation has been demonstrated to exist.

g) The usual complaints of the patient with ileal regurgitation are abdominal pain of various sorts, intestinal gas, constipation, and *nerves*.

The pain complained of may be a slight general abdominal discomfort, or it may be associated with the presence of gas particularly in the cecum, hepatic flexure or sigmoid loop, more rarely in the splenic flexure. A second type of pain in these cases is a high central epigastric pain which often becomes confused with that of gastric ulcer; this particular type of pain is frequently relieved by increase of the costal angle through appropriate developmental exercises. A third characteristic type of pain is a transverse soreness of the abdomen, apparently definitely related to the existence of a spastic transverse colon. A fourth type of pain is that due to distress of the heart following pressure from excessive gas in the stomach, resembling in character the heart pain complained of by the typical neuro-cardiac asthenic patient.

Constipation is almost universal, being usually of the spastic variety which in the majority of cases shows definite improvement under the administration of atropin.

h) Diagnosis is absolute by Roentgen examination. The presence of a majority of the above signs or symptoms should be sufficient to indicate the necessity for a thorough Roentgen examination of the gastro-intestinal tract. This will usually yield more or less negative results if the examination stops short of the administration of an opaque enema, although the previous examination is of value in determining whether the colon is spastic or whether it has become atonic, the latter condition very possibly a later exhaustion stage following the spastic state.

Judging from the relative infrequency with which patients referred to me, often after gastro-intestinal examination by competent roentgenologists, have previously been given an enema, the opaque enema is considered unnecessary; or possibly this omission occurs because, unless it is habitual routine, the administration of the opaque enema is distasteful. There is no doubt, however, that the opaque enema is the most valuable single procedure available today in the study of conditions existing within the colon and at the ileocecal juncture, especially with fluoroscopic observation of the withdrawal or expulsion of the enema at the completion of the usual examination.

In all of the 50 cases here reported, the administration of the opaque enema was followed by a study of the filled colon, the enema was then withdrawn, the entire procedure being followed fluoroscopically. Much colon physiology is learned by watching the colon empty itself. If the terminal ileum is filled by regurgitation, it usually remains filled until the entire colon empties from cecum to ampulla. The ileal contents thereafter may or may not be seen to follow the course of the colonic contents from cecum to ampulla, the whole distance occasionally being traversed by a block of opaque substance previously in the ileum, in the space of a comparatively few seconds. To see a long, ptotic, festooned, loosely attached, M-shaped colon suddenly transform itself into a short omega style colon without evident excess of length anywhere, in order apparently to facilitate the smooth and rapid passage of large blocks of colonic contents, is to get new ideas on whether or not to do frequent fixation of the colon. Fluoroscopic study of the withdrawal of the opaque enema, is certainly too little practiced. There is no substitute for this procedure, which if properly carried out is rapid and satisfactory both to the patient and to the observer.

III. Frequency of Ileal Regurgitation.

On the basis of the observations by Case, Cole, Baetjer, and others, it would seem that ileal regurgitation may be expected in 20 to 30 per cent. of any series of gastro-intestinal cases. Therefore, if one finds a frequency higher than 50 per cent. in any consecutive series of cases, it must be considered distinctly unusual.

In the series of 50 consecutive chronic intestinal invalids upon which this paper is based, the existence of ileal regurgitation was demonstrated in 80 per cent. of the cases. Such a high frequency of ileal regurgitation is, to say the least, surprising, and calls, if possible, for explanation.

The technique employed was identical. All observations were made by both Dr. L. B. Morrison and the writer. A large rectal tube was used. The enema was allowed to flow in until the cecum was barely well filled. The flow of the enema was then checked before any evidence of undue distention occurred. No manipulation was indulged in until it had been determined if the barium suspension would be carried back into the ileum by antiperistalsis. Occasionally a half minute or so would elapse before antiperistalsis became active enough to produce visible ileal regurgitation. Only cases in which antiperistalsis by itself and without external pressure carried the barium back into the ileum, were recorded as having regurgitation. On the other hand, it may be stated that it was rather unusual to find a case in which factors other than antiperistalsis did cause regurgitation, possibly in view of the precaution taken against over-distention of the cecum. Cases showing regurgitation after changes in position or the strains incident to evacuation of the bowels, were not classed as having regurgitation.

After fluoroscopy of this filled colon, and the taking of plates as necessary, the receptacle containing the enema was dropped to the floor and the emptying of the colon was studied fluoroscopically. This procedure yielded much information concerning colon physiology, and cannot be too highly recommended to those interested in gastro-enterology.

In view of the above precautions, there is little doubt about the facts in relation to this 80 per cent. frequency of ileal regurgitation in the group of cases studied.

Consequently, it seems necessary to look for some underlying reason for such frequency, in connection with the condition of the patients themselves. The inference is that this astonishingly high frequency of regurgitation found in these cases may be considered as a factor worthy of attention in the carrying out of any treatment directed toward improving the general health of the patients presenting this condition.

IV. FREQUENCY OF THE EXISTENCE OF "NERVES."

The complaint of *nerves,* sometimes of most bizarre type, seems almost constant in the chronic intestinal invalid. In fact, there was but one case in 50 which did not complain of some form of increased nervous irritability or tension. The frequency of this complaint in the series of cases under consideration is therefore 98 per cent. One example is sufficient to indicate the curious manifestations of *nerves* which these patients may present.

Mr. R. P., sculptor, white, aged 35, single, height 6 feet, weight 205 pounds; with good habits; brought up on a Western ranch. Was first seen October 7, 1919.

Family History.—Father died of cancer of the rectum at 66 years of age. Family history otherwise negative.

Previous History.—Patient lived always in the open, mostly on horseback, until the age of 22. He could always keep up with the others in doing heavy physical labor, but he "always felt as though he weighed a ton," and never liked exercise, feeling that he always expended excessive energy for the results produced. Patient had measles at 8, mumps severly at 14, and scarlet fever at 16. Otherwise, early history negative. Patient began to worry about the future at 12 to 14 years of age, probably on account of the straightened circumstances of the family. He always had a desperate desire to study art, and has been active in sculpture for the last 15 years.

Present Illness.—Nervous, worried easily, gets along but uses up excessive energy. Has never collapsed but frequently has difficulty in controlling his temper, and after the close of any strenuous session with students or other, is apt to get weepy. Has fainted four times, the last two times after being waked up at night by abdominal cramps. These occasions were one year ago and two nights before first coming for advice. On the latter occasion patient waked up with abdominal cramps, promptly fainted, and was not normal for an hour.

Patient complains of a "woozy feeling in the lower jaw," and "a hot spot on the crown of the head roughly circular in shape, about three inches in diameter, approximately in the position of a priest's tonsure." This hot spot has been present for many months, and can be accurately outlined with the finger. The existence of this hot spot has seemed parallel to a decreased efficiency in the power of expression of thought, and other forms of mental concentration. Patient has suffered intermittently from coxalgia, this condition having been almost constant for the past few months.

Physical Examination.—Physical examination in general negative, the patient being tall with a large frame, and very sloping shoulders. The musculature is at present deficient. Posture fair. Skin negative, except for some discoloration, and numerous pimples on the back. Pulse 65, blood pressure 140-90. Urine negative.

Gastro-intestinal Roentgen examination, beginning October 9th, showed a sluggish stomach with slight pyloric spasm, normal duodenum and normally shaped colon. The ileocecal valve was incompetent, the last one or two feet of ileum remaining filled after the evacuation of the enema. The ileum seemed sluggish in action. To promote possible action, the patient was given a half glass of water to drink. Twenty or 30 seconds afterward, the terminal ileum was suddenly and completely evacuated, after two definite and very vigorous forward and backward movements of the block of opaque substance contained in the ileum. It was very striking to observe this sudden activity of the ileum apparently as a result of the ingestion of a half glass of water.*

Treatment.—Active treatment, chiefly by diet and exercise, commenced on October 11, 1919. Improvement was rapid and continuous. On October 25, the patient reported that the hot spot on the back of his head had been gone for 24 hours and that his head was clearer. On October 29th patient stated that there had been no return of the hot spot and that his head was perfectly clear. He said that he had done a hard day's work during which he had not felt tired as usual. He considered himself cured of his *nerves*, and had no more coxalgia. On November 5th patient reported that he was "feeling fine." He stated that he was convinced that an excessive use of food had been a serious factor in his condition, and that he would hereafter be distinctly temperate, both in food and candy. He said that he was very pleased because he had been able to do a real piece of constructive writing for a report, which has been accepted as a unanimous opinion by the committee on which he was working. He also stated that this was the first time for nearly two years, when he had felt that his head was in good order for intellectual purposes. On December 3rd, patient wrote that he was "too well to waste time or money on doctoring."

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*This observation has been repeated, and the procedure outlined has been found useful on numerous occasions since the above was written.

V. Relation of Ileal Regurgitation to "Nerves."

Since *nerves* of some kind were present in 98 per cent. of the 50 cases, and regurgitation was present in only 80 per cent., the *nerves* cannot be said to be always secondary to ileal regurgitation. A study of the 20 per cent. of cases in the series free from ileal regurgitation, gives the following results:

The ten chronic intestinal invalids with *nerves* but with normal ileocecal mechanism were divided between the herbivorous and carnivorous human types[10] in the proportion of 6:4. In the herbivorous group, however, one-half the cases had a definite organic lesion, as a possible cause for nervousness. In the remaining herbivorous cases, and in all of the four carnivorous cases, there was no obvious cause for *nerves* outside the neuro-psychiatric sphere. It is probable that overwork, mental worries, and fatigue were factors in all cases. It is of interest, however, that none of the carnivorous cases had an organic factor. This is directly in line with the results of a study reported by Hodskins[11] concerning human type and the etiology of epilepsy, following suggestions by the writer.

As a result of the above, it seems probable that ileal regurgitation acts, if at all, only as a precipitating factor which promotes possibly through chemical metabolic means, nervous irritability. This is the more probable, in view of the fact that various small forms of superficial nervous irritability disappear rapidly, after the application of a diet directed toward preventing the usual undesirable symptoms of ileal regurgitation parallel with the improvement noted in the functioning of the digestive system. After this comparatively rapid primary improvement, however, there then remains the fundamental irritability of the nervous system, varying with the individual, to be combated by appropriate therapeutic procedures.

VI. Treatment.

Treatment is divided into the early, intermediate and late stages of convalescence. In general, during the early stage, the objectives are external and internal rest, varied by carefully controlled periods of exercise. This means that after perhaps a few days of complete bodily rest, if this is deemed essential, the patient, if as usual ambulatory, takes up again his daily life, but with the work periods broken as nearly as possible by five-minute rests every half hour during the day. This specification is based upon the principle that rest from fatigue is effective inversely according to the duration of the preceding fatigue periods. Internally, rest is obtained by the utilization of the simplest softest bland diet which can be made satisfactory to the patient. At this time, eggs, meat and fish are absolutely contra-indicated as being potentially putrefactive substances. The duration of the early stage of convalescence covers usually from one week to one month.

The intermediate stage covers perhaps the next two months of convalescence. During this period the diet is gradually amplified, and the patient begins to approximate more nearly normal conditions in the manner of his daily work. During this stage, it is important to warn the patient that it is impossible to expect continuous recovery. The usual sequence of events is that the proportion of good days to bad increases, and that the intensity of the undesirable complaints becomes less. As a rule there will be at least two or three opportunities to demonstrate to the patient the fallacy of his allowing himself full liberty of action simply on account of a sensation of well-being, too early in the progress of convalescence. The invariable result of this procedure is subsequent collapse, or at least a definite setback in progress, and there is usually opportunity to prove that a single indiscretion may not be wholly recovered from for as much as one or two weeks. The word indiscretion is applied both to the expenditure of bodily and mental effort, and to the excessive use of undesirable foods.

The late stage of convalescence covers from two or three months to a year or more, since if the patient has properly learned his lesson he may continue to improve in a general way over perhaps two or three years. This is a period of active experimentation on the part of the patient. Having learned his lesson, it is for him to determine how nearly he can approximate a so-called normal standard of work, and then to realize that every excess beyond his own limit must be paid for by a relative return of the symptoms from which he has become free. It is, therefore, for him to determine whether the emergency justifies the sacrifice which he knows must follow.

There are five totally distinct factors of value in treatment. These are grouped under the following headings:

1. Social.
2. Mental.
3. Dietary.
4. Orthopedic.
5. Glandular.

They are discussed at some length in another paper,[12] three paragraphs from which are here quoted.

"The dietary employed is based in part upon a consideration of the large amount of work by many reputable investigators, which tends to prove the ease with which the intestinal flora can be changed by a modification of diet; also, upon a consideration of the fact that ileal regurgitation is an exceedingly common finding in the chronic intestinal type of invalid. Ileal regurgitation was, for instance, demonstrated in 80 per cent. of a consecutive series of 50 chronic intestinal invalids. Eggs, meat and fish, as being potentially putrefactive, are at first absolutely excluded from the dietary, and the use of milk is restricted to the amount ordinarily employed in cooking. After a few weeks of complete exclusion of the above foods, it is very common to find that the patient's skin is distinctly less muddy. Slowly, the yellowish color of the skin is replaced by white and then by pink, a transition which is greatly appreciated by the average woman patient. During this preliminary period of marked restriction of the diet, bran and the coarse vegetables are eliminated. No diet list is given, the patients being thus required to use their own brains in the working out of a suitable dietary. One advantage of this system is that the patient gradually acquires an adequate dietary for which he has the least possible dislike. If the dietary actually employed by the patient is written out and continually checked up at successive visits, the results seem better from the point of view both of interest of the patient and of the physician, than if a diet slip is handed the patient at the first visit. Eggs, meat and fish are rigidly excluded from the diet for at least one month. During the latter part of this month, however, there is an increasing use of egg as a flavoring in puddings or other cooked dishes, in which the egg is finely divided by mixture with the various carbohydrates or vegetables. Meat gravies as relishes precede the use of meats as such. During the month of restricted diet, liberal use is made of two special articles of food, in conjunction with the usual fruits, starches, vegetables and fats. These two articles are, cream cheese (or perhaps the ordinary cheese when used as a flavoring in various cooked dishes) and gelatine. The frequent use of flavored gelatines in the form of salads or desserts is encouraged. The necessary minerals, and the water and fat soluble vitamines, are provided for by the use of raw fruit and green vegetables, and a good grade of butter. In this connection it is surprising to discover how few people are acquainted with the virtues both from a financial and an economic point of view, of chopped raw cabbage. This food as a salad with French dressing has been taken time and time again without the slightest discomfort or after effects by patients who had previously stated that they could not possibly eat such a coarse vegetable.

At the end of a month or six weeks, if the progress of the patient justifies it, as evidenced by decreased constipation, lessened irritability, lessened disturbance from gas, better appetite and sleep, better color of the skin and fewer headaches, meat is put back into the dietary at first once a week. The intervals between the days on which meat is used are thereafter shortened according to circumstances. But in the chronic intestinal invalid it has been found most unusual, if meat, eggs or fish as such can be handled with comfort more frequently than two or three times a week. The intermittent use of eggs, meat and fish has another virtue in addition to its economy and its tendency to discourage the growth of undesirable intestinal flora. If the patient has only to wait over one or two days of restricted diet before being allowed a day of full diet, there seems to be much less desire for large quantities of these potentially putrefactive foods, which are handled with difficulty certainly in all patients with demonstrated ileal regurgitation.

In practice, on completion of the history, physical examination and the routine Roentgen examination of the gastro-intestinal tract, of which fluoroscopic study of an opaque enema is an essential element, the social and mental factors receive first consideration in the progress of treatment. As soon as these factors are, so to speak, stabilized, the diet is taken under serious consideration. In conjunction with diet, frequent use is made of mineral oil, agar, yeast and atropine, and every attempt is made to take advantage of habit formation and the natural rectal reflexes. As soon as the diet has become satisfactory, the application of orthopedic procedures follows, and finally comes the use of the glandular preparations when indicated. Thus, by the end of the first month or so, all five of these totally independent factors are being simultaneously employed in the effort to bring about as rapidly as possible, an improvement in the general condition of the patient. Belts are rarely used, since they tend to decrease the faith of the patient in his own ability to carry himself through his day's work. Only slight attention is paid to the question of weight, provided there is not a progressive loss in weight. Not infrequently one may find an apparently healthy person of almost incredible thinness. On the other hand, excessive fat is no guarantee of health. Usually, however, the very thin patients will put on at least from 10 to 15 pounds in the course of a few months without apparent effort, if otherwise satisfactory progress is being made."

Case,[13] who has devoted serious consideration to this question, has stated that if the ileocecal mechanism is once incompetent, it is always incompetent. This is doubtless true in the majority of cases, but not always, as indicated by a case reported by the writer,[14] in which re-examinations at the end of one and two years, of a patient with previously demonstrated ileal regurgitation, proved that the regurgitation was no longer present. Kellog,[15] one of the few persons in this country who has taken ileocecal incompetence seriously, has even advised operation[16] for the restoration of the ileocecal mechanism. Without discussing the actual merits of this procedure, it may be stated that in practically all cases, satisfactory results in the way of treatment have been obtained by the writer by non-surgical procedure. Numerous cases have been re-examined at various intervals after treatment.

If the ileocecal mechanism is still found deficient, it has at least been found to be much less so than at the original examination, and there has also been observed in every case a striking improvement in the general tone and action of the colon. One may therefore say in regard to operative treatment, that it is usually unnecessary, since satisfactory results can be obtained without it, and in the absence of the risks involved by operation.

VII. RESULTS OF TREATMENT.

Application of the method of treatment outlined above, has yielded the following results in the consecutive series of 50 chronic intestinal invalids upon which this paper is based.

No improvement	6 per cent.
Improvement	8 per cent.
Improvement plus	86 per cent.
Total improved	94 per cent.

In the group of three cases constituting the six per cent. considered not improved, is included one case with ileal regurgitation and concomitant diabetes. Treatment was practically refused for fear of upsetting the diabetic treatment. With this case excluded, the percentage of cases in which no improvement in the general condition was obtained, is obviously lower than six per cent. On the other hand, the obtaining of a marked or satisfactory degree of improvement in 86 per cent. of 50 cases, makes it possible to speak with some assurance concerning the prognosis, when first asked about this matter by a patient who may be in a state of mental perturbation as a consequence of frequent previous medical failures.

On the basis of the above results it is justifiable to tell the chronic intestinal invalid that he has a better than 90 per cent. prospect of improvement, and a better than 80 per cent. prospect of being very much improved as a result of a few months of non-surgical treatment. This applies to the cases in which ileal regurgitation is proven. For the chronic intestinal invalid without ileal regurgitation, progress should be proportionately more certain and more rapid.

VIII. SUMMARY.

1. Attention is drawn to the fact that responsible physicians refuse to become interested in a morbid condition with a history dating back to the Middle Ages.

2. The conditions associated with or resulting from ileal regurgitation do not favor optimum functioning of the gastro-intestinal tract.

3. The patient with ileal regurgitation usually suffers from an increased intensity of any nervous irritability to which he may be liable, during the continuance of untreated ileal regurgitation.

4. Immediate treatment of ileal regurgitation is by simple, bland, non-putrefying diet.

5. Adequate treatment must consider every aspect of the patient's welfare.

6. The results of treatment are indicated.

IV. Conclusions.

1. Ileal regurgitation is not a normal condition. Disregard of its existence does not promote health.

2. The existence of ileal regurgitation may be demonstrated with ease and certainty.

3. In unselected gastro-intestinal material, a frequency of 20 to 30 per cent. of ileal regurgitation is to be expected. In a consecutive personal series of 50 chronic intestinal invalids, the frequency of ileal regurgitation was 80 per cent.

4. Marked improvement in the general condition of 86 per cent. of this series of chronic intestinal invalids, resulted from treatment directed in part toward relief from the results of the ileal regurgitation itself, and directed in part toward improving the general condition of the patients.

5. With easy diagnosis assured, and the possibility of obtaining satisfactory results by non-surgical treatment sufficiently demonstrated, the physician who prefers to continue to ignore the existence of ileal regurgitation may expect to have his patients suffering from ileal regurgitation take matters into their own hands, if they find themselves continued in a medieval status of medical neglect with regard to progressive treatment of their malady.

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DISCUSSION.

Dr. Allen Jones, Buffalo: That ileal regurgitation takes place seems quite well assured. How often it takes place it is not only difficult but impossible for us to estimate, because we are apt to get a false impression from our chief means of diagnosis, which is radiography. Dr. Bryant has, however, eliminated in a large measure the objections so often made to this method of diagnosis, the objection being that filling the colon with a heavy substance like barium under pressure is, in itself, sufficient to cause regurgitation of some of this material into the ileum. As you will recall, in Dr. Bryant's paper, he particularly avoided increased cecal pressure, allowing, as viewed by the fluoroscope, the barium to be brought back by retroperistalsis into the cecum, and there be regurgitated into the ileum. Whether this is in itself a pathological condition of great importance or not, may also be discussed. If the factors of safety in the gastro-intestinal tract remain unimpaired—that is, if the patient has a perfectly healthy epithelial lining in the ileum—is it necessarily a very deleterious thing if, for a short time, some of the contents are regurgitated from the colon? In a paper read in Buffalo, some years ago, at the Buffalo meeting of the State Society, in discussing a question involving intestinal toxemia, I said that contents, once having entered the colon, would probably play nothing but a harmful rôle if regurgitated again into the ileum. The question is a very large one, and one upon which much difference of opinion has existed among clinicians, and probably will exist for a long time, some believing but little of so-called intestinal intoxication, and others making a great deal of it. We cannot positively state, however, how often or when ileal regurgitation occurs in the ordinary course of life. It would seem to me very desirable to give small masses of bismuth with the ordinary meals, or else to swallow with a meal small bits of metal shot, which, round and smooth, would do no harm; and to follow these and see whether, with the food going through, they actually regurgitated and went back and forth from the cecum to the ileum, and from the ileum to the cecum. Perhaps this has been done, but I do not know of it. It seems probable that the radiographers may find some method of practically determining this question.

So far as the causes of ileal regurgitation are concerned, and its surgical treatment, it would seem to me that surgery is chiefly justified in cases in which it will relieve the cause of regurgitation; as, for instance, chronic appendicitis, or any condition which mechanically pulls on the ileum, pulling it away from the cecum. But it would seem to me rather dangerous to carry out a procedure which may really cause a more serious condition, a narrowing of the ileocecal opening and real ileal stasis, which would be more serious than ileal regurgitation.

So far as the treatment outlined by Dr. Bryant is concerned, I am entirely and heartily in accord with him. Not a little depends upon the pertinaceous, persevering plan of medical and orthopedic measures and therapy which he insists upon, and on the diet, carried out faithfully with these other measures in his cases, which have resulted in a very large percentage of cures. We are indebted to the doctor for bringing this important subject before us.

Dr. Franklin W. White, Boston, Mass.: I should be very glad to ask Dr. Bryant two questions. One is whether he has considered the possibility of variation in the presence or absence of ileal regurgitation at different times. He knows that at the present time we are very likely to base our diagnosis or our estimate of a case on a single Roentgen examination, naturally; because these examinations are expensive, elaborate things. I wondered whether he had considered the possibility that this ileocecal valve, which is really not so much a valve as a sphincter, might at some times be willing to open and let things through, and at other times not, just like the cardia or the pylorus.

Another thing that I wanted to ask is whether, in the cases that improved under treatment, there was any relation to the degree of ileal regurgitation present—whether the ones with a great deal of regurgitation were decidedly a more difficult group to deal with.

A third question is whether any effort was made to check up the results in the cases that had done very well indeed—whether the regurgitation had disappeared or was pretty well out of the field.

Dr. Bryant (closing): I assume that if a person is not very well, ileal regurgitation is a potential factor for trouble, and I believe that it is one added factor in preventing recovery if the patients get into a tight place, since they are always short of energy. It takes 98 per cent. of what energy they ordinarily have, for the common processes of living. If something happens in the family that upsets their routine and causes more work for them, they have not the reserve energy to stand it. The patients tell me that after treatment they can get along and do their work infinitely better than they could before treatment.

The question of the function of the ileocecal valve is very interesting. I do not know how many observations there are on it, but MacEwen had a case 15 years ago, which he followed for months, in one of the Scotch Hospitals. Through the open stoma, he was able to observe that the natural method of discharge of the fluid intestinal contents into the cecum, was by rhythmic spurts, with a quiescent period intervening. The rhythmic period was apparently that more or less fundamental to the intestine, and the spurts continued until the food in the terminal ileum had been evacuated. Then the valve would remain shut for a long time. It would almost always open again, with a recurrence of the spurts within two minutes after putting food in the stomach. MacEwen had no X-ray available, but it was on account of his observation that I gave my patient a glass of water, to see whether his pyloric reflex, the existence of which was proven by Elliott, would work. It did. I do not see how you can assume that if the ileum is an important absorptive area, it can be desirable to have decomposition products in the ileum. Unless one can devote some 20 to 30 hours to it, it is scarcely worth while to attempt this form of treatment, but almost anyone who is willing to give the necessary 20 or 30 hours per patient, can duplicate the figures that I have given.

The glandular cases I did not mention. One was a case in which there was a definite connection between the marked suprarenal insufficiency and the general condition of the patient, who was a woman. She had exhausted her failing strength by going to a party and dancing hard. She collapsed, vomited almost constantly and was unconscious for over a week. She was almost a chocolate-brown color all over, and the circulation in her arms from her elbews down had, judging from her cold marbled cyanotic forearms and hands, all but ceased. She was given about 30 grains of suprarenal whole substance for many weeks. It may not have had any effect, but the circulation improved, color and warmth extending from elbow to finger tips at the definite rate of four inches a week. The brown color of the skin disappeared as the circulation returned, but more slowly.

In regard to the question that Dr. White asked, as to whether there was any error in observation, I would say that if 80 per cent. open in ordinary technique—

Dr. White: That was not my question. It was whether, in a particular case, you had any way to check up whether the valve was open at one period and found entirely closed when observed again.

Dr. Bryant: You might miss incompetence if the valve or sphincter were temporarily closed, but in these cases it was under observation from three to five minutes. It does not infrequently appear to be tight in the first couple of minutes. Then with increased antiperistalsis, regurgitation becomes evident.

As to the question of the relation between the degree of regurgitation and the symptoms, I would say that this has been remarked upon by a number of observers. There is a correlation between the speed of improvement and the degree of ileal regurgitation. Case states that if the valve is once open, it is always open. That is not entirely true, because I had one case which became normal as proved by X-ray. I have had two cases in which there seemed to be a definite traumatic origin for the regurgitation. Both patients were lifting heavy weights, had severe right lower quadrant pain, and suffered from severe prostration for two weeks afterward. One was an elderly man with a wide-open valve. He was fluoroscoped one year later, and not even violent pressure would open it. He was fluoroscoped after one, two and three years, and it never was found to be open again. I have seen other cases in which the valve remained open, but in which a much less degree of regurgitation was found on reexaminations. In these cases there was a coincident improvement in the tone of the colon at each successive fluoroscopic examination.